VOLUME 36 NUMBER 3 MARCH, 1953

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OF
OPHTHALMOLOGY

THIRD SERIES FOUNDED BY EDWARD JACKSON

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DEPARTMENTS

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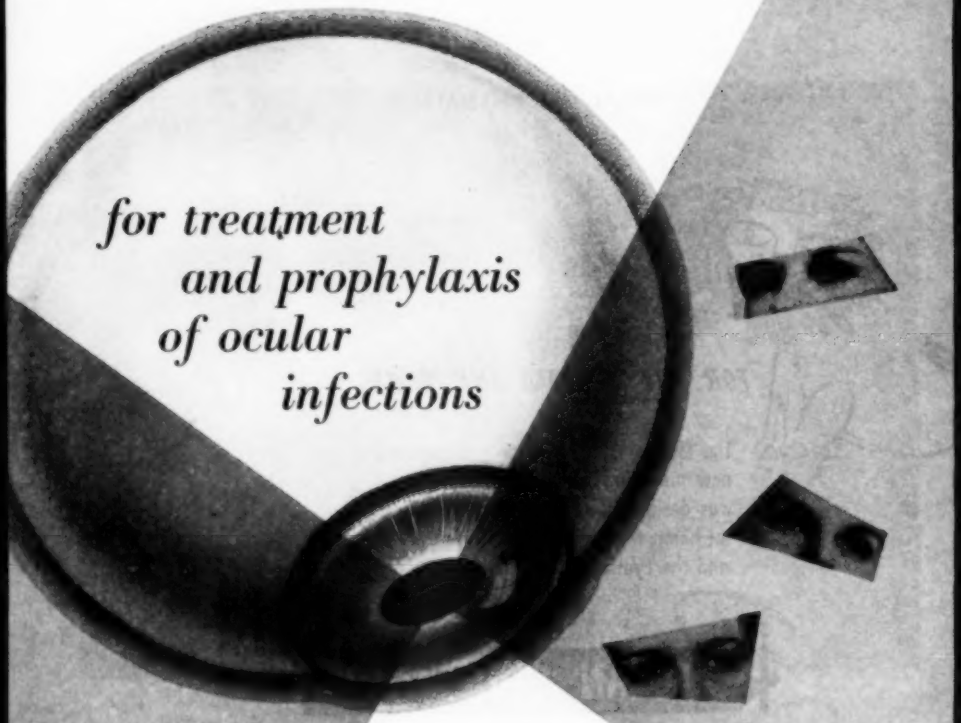

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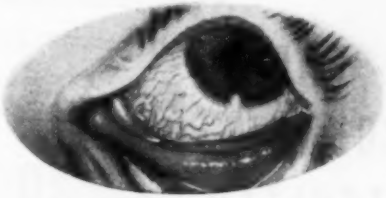

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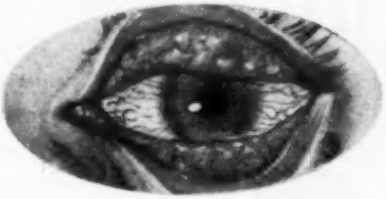

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1. *Wolman, B., and Holzel, A.: Brit. M. J. 1:419 (Feb. 23) 1952.*
2. *Mitsui, Y., et al.: Antibiotics & Chemotherapy 1:253 (July) 1951.*
3. *Douvas, N. G.; Featherstone, R. M., Braley, A. E.: Arch. Ophth. 46:57 (July) 1951.*

Note: The cases reported above are based on work with the original Terramycin Ophthalmic preparations — before the addition of Polymyxin B Sulfate for even greater therapeutic effectiveness.

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From
DRUG TRADE NEWS
February 2, 1953

Ophthalmic Drug Sterility Rulings Released By FDA

Say Investigations Reveal Contaminated Products Have Produced Injuries

WASHINGTON — New sterility regulations have been issued by the Food and Drug Administration here, for antibiotic solutions. Preparations under the revised regulations are penicillin-streptomycin opthalmic, neomycin opthalmic, ophthalmic, and bacitracin opthalmic.

The order, carried in the Federal Register for Jan. 1, requires that these preparations be sterile and allows for the addition of more suitable harmless preservatives in the production of these compounds.

Provide For Dating

The new regulations require that if these drugs are packaged with a container of a diluent, the diluent must be sterile distilled water, U.S.P.

It is pointed out that investigations by pharmaceutical manufacturers, physicians, and the FDA have revealed that liquid preparations for ophthalmic use that have been contaminated by viable organisms have been responsible for serious eye injuries, and, in some cases, complete loss of vision.

Purity Defined

As a result, the FDA has concluded that preparations designed for opthalmic administration fall below their standards of purity and quality and may be unsafe for use if they are not sterile.

Accordingly, the FDA says, liquid preparations offered for or intended for ophthalmic use which are not sterile may be regarded as adulterated within the meaning of Section 501 (c) of the Federal Food, Drug, and Cosmetic Act and also may be misbranded within the meaning of Section ... Act.

From AMERICAN JOURNAL OF OPHTHALMOLOGY
June, 1952

PRESCRIPTION OPTICIANS

AMERICAN JOURNAL OF OPHTHALMOLOGY

SERIES 3 · VOLUME 36 · NUMBER 3 · MARCH, 1953

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Frontispiece illustrating article by F. Necdet Sezer

FIG. 1

FIG. 2

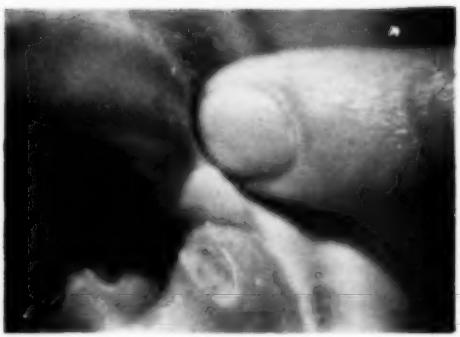

FIG. 3

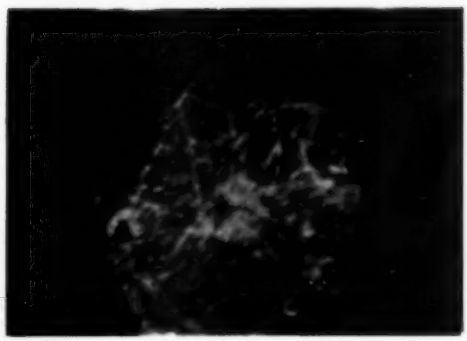

FIG. 4

FIGS. 1 TO 4 (SEZER). (FIG. 1) THE EYE IN BEHCET'S DISEASE. (FIG. 2) THE TONGUE IN BEHCET'S DISEASE. (FIG. 3) THE LIPS IN BEHCET'S DISEASE. (FIG. 4) THE LESIONS IN THE CHORIOALLANTOIC MEMBRANE CAUSED BY THE VIRUS.

AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 36 MARCH, 1953 NUMBER 3

THE ISOLATION OF A VIRUS AS THE CAUSE OF BEHÇET'S DISEASE*

F. NECDET SEZER, M.D.
Istanbul, Turkey

In 1937, Behçet,[1,2] a Turkish dermatologist, first identified as a distinct entity a disease presenting the three cardinal symptoms of iridocyclitis with hypopyon, aphthous lesions in the mouth, and ulceration of the genitalia. Further observations and new cases were added to those of Behçet by Weekers[3] and Franceschetti[4] in 1938, by Wise and Sulzberger,[5] Jensen,[6] and Mach, Babel, and Naville[7] in 1940, and by G. Meyer[8] and Babel[9] in 1943. The disease was first referred to in the medical literature as "Behçet's triple symptom complex" and later as "Behçet's disease" or "Behçet's syndrome."

Actually this was not the first time the disease had been observed. Prior to Behçet's studies, its manifestations had been described by Jacobini[10] in 1894, Sutton[11] in 1895, Neuschuler[12] in 1898, Reis[13] in 1906, Bluthe[14] in 1908, Koppe[15] in 1917, Gilbert[16] in 1921, Weve[17] in 1923, A. Fuchs[18] in 1926, Carol[19] in 1928, and Adamantiadis[20] in 1931.

All these observers, however, considered the symptoms in the mouth, eyes, and genitalia as complications of certain well-known diseases such as tuberculosis, syphilis, rheumatism, and staphylococcal allergy, rather than as manifestations of a morbid entity, and it was Behçet who first defined them as an independent disease probably caused by a virus.

CLINICAL SIGNS OF BEHÇET'S DISEASE

Behçet's disease has three basic and invariable signs common to all cases. These are: (1) Iridocyclitis with hypopyon; (2) aphthae in the mucosa of the mouth; and (3) ulceration of the sexual organs. These will be discussed in order, as follows:

1. *The characteristic ocular lesions* are the most important manifestations and are recurrent, crises developing at intervals of from one to two months over a period of years. Both eyes always have the disease, sometimes simultaneously, sometimes separately. It is the iridocyclitis with hypopyon which attracts the immediate attention in the crisis. The vitreous is also cloudy at this time.

On the basis of our studies of 32 cases in the Clinic of Ophthalmology of the University of Istanbul, however, it is our conviction that the ocular lesions actually begin in the retina and optic nerve and that the uveal disease manifests itself later.

At a very early stage there is a narrowing of the vessels of the retina and there are usually signs of periarteritis and endoarteritis. In the acute phases retinal hemorrhages not infrequently occur. In one case we observed an hyphema in the anterior chamber.

These acute symptoms disappear in from five to 10 days, leaving very few traces, but the recurring crises result ultimately in blindness, either from atrophy of the optic nerve or phthisis of the globe. This end result develops in some patients only after many years and in others in a very short time (fig. 1).

2. *The signs in the mouth* are classical and typical aphthae can be observed in all the parts of the mouth mucosa and on the tongue. During a crisis the patients find great difficulty in eating or drinking (figs. 2 and 3).

* From the Ziya Gun Institute for Research in Trachoma, University of Istanbul.

3. *The manifestations in the sexual organs* are superficial ulcerations. In males they occur particularly on the skin of the scrotum; in females they are found on the large and small labia and sometimes on the clitoris. They resemble the Lipschütz ulcer and are very painful.

In addition to these three major manifestations, various other signs and symptoms may accompany the disease. The most important of these is erythema nodosum which appears in association with a high fever. The nodules tend to be small, few in number, and cyanotic in color. They are painful when pressure is applied and disappear in from 20 to 30 days without leaving any trace.

Other possible associations are furunculosis, pyoderma, abscesses, signs of papulopustular dermatitis, rheumatismal pain and swelling, particularly of the ankles, orchitis, hydroceles, and phlebitis. The patients are usually anemic, complain of headache, and show an extraordinary hypersensitivity and nervousness; some have shown signs of meningo-encephalitis.

The most important characteristic of the disease is its frequent recurrence and the appearance in each recurrence of all three symtoms, either all together or one after the other. Although the intervals between the recurrences vary with each case, their duration is usually about a month.

As the disease progresses the intervals between attacks lengthen until there are finally no further attacks. The manifestations in the mouth and genitalia may continue to appear even after the eye has become phthisic, but if the disease is not complicated by a meningo-encephalitis, it recovers completely by itself after a 15- or 20-year period except, of course, for the blindness.

The onset of Behçet's disease is usually between the ages of 15 and 40 years and most of the victims are men. Infections have not been observed in more than one member of a family.

Etiology

Prior to Behçet's investigations, most workers incriminated tuberculosis, syphilis, focal infections, or allergy as the pathogenic factors in this disease. In the cases of Cavara,[21] Withwell,[22] and Adamantiadis,[20] the Wassermann reaction was positive, but none of the patients improved on specific antisyphilitic therapy.

A number of ophthalmologists, notably Urbanek,[23] suggested tuberculosis, but no specific lesions have been observed in any of the histopathologic examinations that have been made.

Weve[17] explained the manifestations in the eyes, skin, and mucosa as a result of staphylococcal allergy. The finding of staphylococci in blood cultures from certain cases induced a number of workers to accept Weve's suggestion, but the disease continued to recur in spite of anti-allergic treatment.

With regard to focal infections, no positive results were obtained either from the extraction of decayed teeth (Withwell) or from appendectomy (Franceschetti).

Behçet proposed that the disease was caused by a special virus, although he was unable to demonstrate one. The results of his histopathologic and bacteriologic investigations of the aphthous lesions and of his experiments on animals were consistently negative, but he insisted that he had observed inclusionlike forms in smears from the hypopyon of the anterior chamber and from the aphthae.

The most interesting animal experimentation was that carried out in 1945 by Alm and Oberg[24] who were able to produce retinitis, uveitis with hypopyon, and meningoencephalitis in rabbits through four generations.

Isolation of a virus from Behçet's disease

In an effort to carry forward Behçet's line of investigation, more than 30 patients with the disease have been studied in the Clinic of Ophthalmology of the University of Istanbul. Attempts to produce an experimental disease in the eyes of rabbits with the material from the anterior chamber or aphthae

of these patients were unsuccessful. This, and the fact that the eye signs apparently begin in the retina and optic nerve, suggested the advisability of studying deeper tissues. The observations on three patients who were examined with this in mind are described in the following paragraphs.

Case reports

Case 1

C. U. The patient stated that about nine months previously aphthae had begun to appear in his mouth which was very painful when he ate, or chewed, and that a month later scars appeared on the skin of his testis. At the same time his left eye started to ache and his vision became cloudy; four months later the same symptoms developed in his right eye.

Examination of the patient's left eye revealed blepharospasm, ciliary congestion, irregularity of the pupil, pupillary occlusion, blood vessels on the iris, and hypopyon in the form of a line in the anterior chamber. The vision was zero and ocular tension was 30 mm. Hg (Schiøtz).

Examination of the right eye revealed congestion and an irregular pupil which was in a state of miosis. The vitreous was cloudy and the fundus could not be seen. The vision was finger counting at half a meter.

There were various small aphthous lesions in the mouth and on the scrotum and signs of numerous tiny papulopustular lesions on the legs and arms. Both ankles were swollen.

Fifteen days later the aphthae in the mouth and on the scrotum had increased in number and a hypopyon had formed in the right eye. The patient complained of terrible pains in his left eye and was highly agitated.

An enucleation of the right eye was performed.

Case 2

F. S. In 1940, the patient had an illness with a high fever which resembled malaria. His fever went up to 39.5° to 40°C. and then decreased with accompanying profuse perspiration. This feverish period lasted for a week and was followed by a calm period lasting from two to three weeks.

After this period of remission another similar crisis occurred. After two years of recurrences, the left eye began to be congested and cloudy. Since the patient's intradermal tuberculin reaction was positive, he was subjected to tuberculin treatment for one and a half months.

During this time, his right eye also began to ache and aphthae appeared in his mouth and on his scrotum. The crises which had formerly recurred once in three weeks now recurred only once in two months. The patient stated that up to the time of examination he had had 60 crises.

Examination of the right eye revealed signs of iritis, atrophy of the optic nerve, and diminution of the retinal arteries which showed periarteritis and thrombosis. In the retina were large and small foci of chorioretinitis. The vision was zero.

Examination of the left eye revealed ciliary congestion in the bulbar conjunctiva and a hypopyon in the form of a line in the anterior chamber. The pupil was irregular, the vitreous cloudy, and the optic nerve dark red with its boundaries unclear and edematous. The retina was swollen and detached around the optic nerve, resembling the chemosis in the bulbar conjunctiva, and was yellowish in color. Vision was reduced to light perception.

The subretinal serous exudate obtained by puncture of the eye of this patient was used as material for laboratory experimentation.

Case 3

K.S., a woman, aged 30 years, said that six months prior to examination her left eye began to ache and became cloudy. She recovered from this attack but the symptoms kept recurring. At the same time aphthae appeared on her tongue and genitalia.

On examination, the right eye was found to be normal. In the left eye there was ciliary congestion. The anterior chamber had a slight and narrow hypopyon. The pupil was irregular and there were posterior synechias. The vitreous was very cloudy. The fundus could not be illuminated. Vision was reduced to light perception. Ocular tension was 40 mm. Hg (Schiøtz).

Various aphthae had developed in the mouth and a wide ulcer had appeared on the small labia.

A subretinal serous fluid was obtained from this eye by puncture and was used for studies of the virus.

Histopathology of Case 1

The enucleated right eye from our first patient was sent to Dr. Michael J. Hogan of the Department of Ophthalmology of the University of California School of Medicine in San Francisco for histopathologic examination. The results of his examination are given below in his own words taken from his report:

"Circumcorneal tissues. The conjunctival epithelium is almost entirely missing; only the basal layer is present. The stroma shows some evidence of degeneration with edema and round cell infiltration.

"Cornea. The corneal epithelium is also almost entirely missing. Bowman's membrane is intact but at the periphery there is some degeneration as a result of the long-standing inflammation. The corneal stroma shows separation of the fibers by edema fluid and infiltration of many inflammatory cells. Descemet's membrane shows some areas of

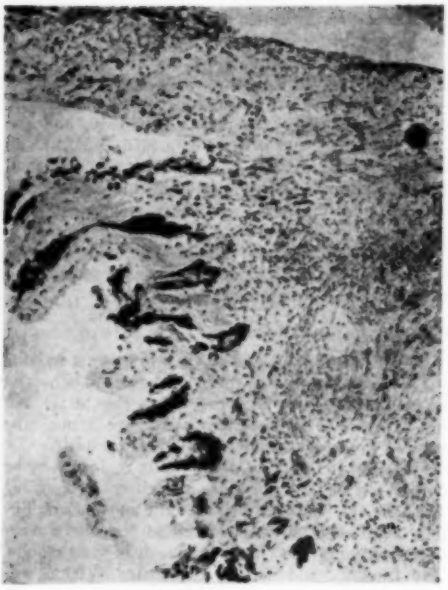

Fig. 5 (Sezer). Ciliary body.

folding. Occasional lymphocytic keratic precipitates adhere to the cornea.

"*Limbus.* Schlemm's canal is patent. The deep scleral vessels show perivascular inflammation. The trabecular meshwork shows edema and round-cell infiltration.

"*Sclera.* The portion of sclera which is attached to this eye shows no gross abnormality.

"*Anterior chamber.* The angle is open. The chamber is extremely shallow due to forward protrusion of the iris. There is a moderate amount of serous exudate in the angle.

"*Iris.* The iris shows evidence of a long-standing inflammation with extensive atrophy of both the stroma and the pigment layers. The entire iris has been affected by the inflammatory process and there is a moderate, diffuse, round-cell infiltration throughout. A thin fibrovascular membrane covers the anterior surface. There is a very dense pupillary membrane. The epithelium is almost entirely atrophied.

"*Ciliary body.* The ciliary body shows extensive destruction of both the epithelial layers and the stroma. The muscularis fibers are separated by edema fluid and round-cell infiltration. The processes show slight hyalinization. Considerable inflammatory exudate lines the inner surface of the ciliary body. The exudate is composed mainly of serofibrinous fluid which contains many round cells and some hemorrhage. As one approaches the limbal region there is a very intense inflammation with marked destruction of the posterior ciliary body and peripheral choroid. The ciliary body and the choroidal fibers are necrotic and contain hemorrhagic exudate as well as chronic inflammatory cells (fig. 5).

"*Choroid.* The choroid also shows marked inflammation with edema of both the suprachoroidea and the choroidal stroma. The epithelium shows marked atrophy.

"*Retina.* Most of the retina is missing from this eye.

"*Optic Nerve.* The optic nerve shows marked inflammation of the disc with necrosis of the nerve fibers and glial bundles on the surface of the disc. The optic nerve fibers show extensive atrophy. The vitreous

Fig. 6 (Sezer). Optic nerve.

and lens are missing from the eye (fig. 6).

"*Diagnoses.* (1) Uveitis, Behçet's disease. (2) Occlusion and seclusion of the pupil. (3) Papillitis.

"*Comment.* The main feature of this eye is the severe necrosis which has occurred in the ciliary body and peripheral choroid. The changes which were observed are of a nonspecific character. No specific organisms can be made out in oil immersion views of the microscopic sections."

These histopathologic findings confirm our clinical observation that the disease causes inflammation of the optic nerve.

Cultivation and serial passage of the virus

For the cultivation of the virus, the vitreous taken from the enucleated eye of the first case, cloudy with exudate and blood, and the subretinal serous fluid collected from the eyes of the second and third cases, were inoculated into the chorioallantoic membranes of fertile eggs in three series of experiments.

Method

The fertile eggs were first incubated for 10 days in an ordinary incubator and the positions of the embryos marked by illumination in a dark room. The marked area was then lifted with an electric drill and the shell membrane punched, the chorioallantoic membrane under the shell being left untouched.

A second hole was opened to the air sac of the egg, and through this second hole the air was withdrawn by a rubber nipple; this detached the chorioallantoic membrane from the shell membrane which was also picked away by the same method without damaging the chorioallantoic.

Three-tenths cc. of the material obtained from the diseased eye was inoculated into the exposed chorioallantoic membrane, and the opened shell closed with Scotch tape. The inoculated eggs were then incubated in a bacteriologic oven at 37°C.

Cultural passages of material from Case 1

Progress notes on the experiments performed with this material are summarized in the following paragraphs.

December 22, 1951

The fluid containing blood and exudate obtained by puncture of the vitreous body of the enucleated left eye of the patient was inoculated immediately into eight chorioallantoic membranes.

December 24, 1951

Four of the embryos were dead, four alive. There was no apparent lesion in the chorioallantoic membranes but all of them were edematous. These four membranes were extirpated and ground, and 0.3 cc. of this material was inoculated into eight more chorioallantoic membranes.

December 26, 1951

Four of the embryos of the new series were dead, four were alive. In three of the living embryos there was extensive edema in the chorioallantoic membranes. In the fourth, various large, dense, nonelevated white spots were observed in certain areas; the whole membrane was covered with tiny hemorrhagic spots (fig. 4).

December 27, 1951

Numerous hemorrhagic spots had formed on three membranes and there was a large necrotic lesion in the center of one membrane. All of these membranes had previously been only edematous. The four membranes with lesions were ground and the material obtained was inoculated: (a) into the chorioallantoic membranes of eight eggs, and (b) into the allantoic cavity of four eggs.

December 29, 1951

Four of the eggs whose chorioallantoic membranes had been inoculated were dead;

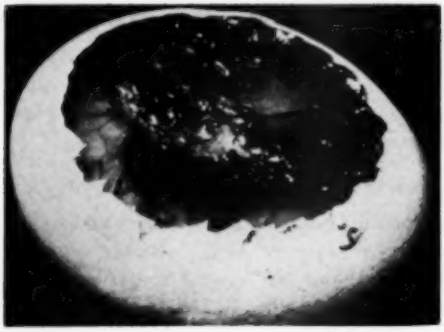

Fig. 7 (Sezer). First lesions in the egg.

four were alive and had developed lesions (fig. 7). These membranes were ground and 0.02 cc. of the material was inoculated intracerebrally into 10 white mice, three weeks old.

January 3, 1952

Roughening of the coat, agitation, tremors, and at last paralysis of the hind legs were observed in two mice. The brains of these mice were extirpated and ground under sterile conditions. Suspensions were prepared containing 1:10, 1:50, and 1:100 of the brain material, and 0.03 cc. of each of these suspensions was injected into the brains of eight mice.

January 12, 1952

Manifestations of encephalitis were observed in the group of mice to which injections of the 1:10 dilution had been given and seven of them were dead on January 16th. The brain of the last mouse, which was about to die, was extirpated.

After five days, paralysis began in the mice which had received the material in the 1:50 dilution; the paralysis had developed in six mice by the seventh day, and subsequently in the other two. By the 10th day, six of the mice were dead and the brains of the remaining two were extirpated; the hind leg of one of these was bluish and gangrenous.

In the mice which had received the 1:100 suspension, strong manifestations of encephalitis were observed in four and weak manifestations in the remaining four. The brains of the four mice showing strong signs were extirpated. Of the remaining four, one died in 10 days, and the brains of two others, which were about to die, were extirpated. One recovered.

From the last group, then, which had received the 1:100 dilution, six brains were extirpated and ground. Suspensions in a 1:100 dilution of this material were prepared and inoculated as follows: (1) 0.1 cc. into the vitreous of two rabbits, (2) 0.02 cc. into five mice intracerebrally, and (3) 0.02 cc. into the chorioallantoic membranes of 10 eggs.

Material from the typical lesions formed on the chorioallantoic membranes of five of these eggs was inoculated into 10 more eggs in a 1:10 dilution. In three passages thus prepared, five of the 10 eggs died; in the remaining five, lesions were formed; but in the fourth passage the death of the embryos increased to eight of the 10.

A 1:100 suspension was therefore prepared. With this dilution death occurred in seven of 10 and the dilution was again changed to 1:1,000, and this concentration was maintained up to the 20th passage. At this passage the lesions regressed, and 1:100 suspensions were again used. The infective agent is now being maintained in its 30th passage (fig. 8).

Cultural passages of material from Cases 2 and 3

Experiments with material from the second and third cases of Behçet's disease followed approximately the same pattern of development as outlined above for Case-1 material except that in these passages the original membrane was not passed from the brain of a mouse as in the first case but was followed on the chorioallantoic membrane only.

Cultural passages of the virus in the allantoic fluid

After a primary passage on the chorioallantoic membrane, the original material from

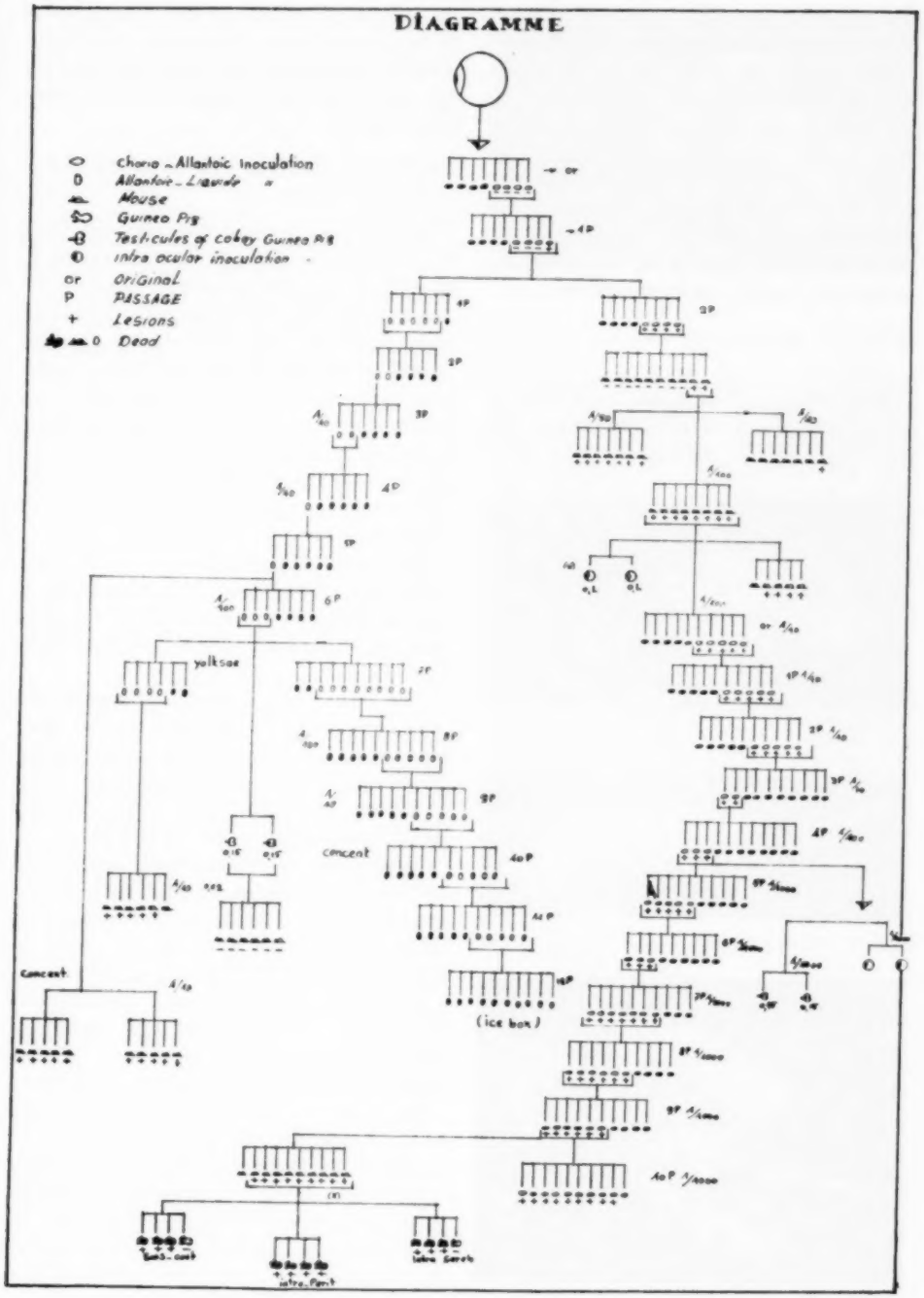

Fig. 8 (Sezer). Diagram, showing passages of infective agent.

all three cases was injected into the allantoic cavity of an egg and passages were obtained by the cultivation of the virus in this fluid.

In the course of these passages the allantoic fluid was injected into the brains of mice, which later manifested typical encephalitis and died. It was thus apparent that the virus could be propagated in this fluid, a fact subsequently confirmed by the infected fluid's proving to be a perfect antigen in complement-fixation tests.

Cultural passages of the virus in the yolk sac

The infected allantoic fluid was also inoculated into yolk sacs and passages were made through this channel. Suspensions of the yolk-sac membrane produced encephalitis in mice and typical lesions on the chorioallantoic membrane, indicating that the yolk sac was a favorable medium for the passage of the virus.

Fig. 9 (Sezer). Paralysis of the mouse.

Fig. 10 (Sezer). Clonic convulsions of the mouse.

Fig. 11 (Sezer). Tonic convulsions of the mouse.

Passages of the virus in various laboratory animals

Mice. Since the material obtained from the eyes of the three cases was limited in quantity, it was not possible to inoculate the original material directly into the brains of mice. However, we were able to produce typical encephalitis in mice by brain inoculation with viral material obtained from lesions formed on the chorioallantoic membrane, from allantoic fluids containing the virus, from suspensions of yolk sac membrane, and from exudates from the eyes of infected rabbits.

The manifestations observed in the mice were roughening of the coat, inactivity or hyperactivity, tremors, circling, hunched position, stiffening of the tail, and paralysis (fig. 9).

The "spin test," consisting of rotating the mouse one way, then the other, while holding him suspended by the tail, then watching his reaction after release, was frequently a valuable procedure. It induced rolling or clonic or tonic convulsions (figs. 10 and 11).

After showing these signs the mice died in two or three days. In mice which had been inoculated with small doses, these manifestations were temporary. Those that survived lost the hair on their backs (fig. 12).

Some mice showed signs of thrombophlebitis in the fore and hind legs; the whole leg became swollen and assumed a dark bluish color. This manifestation was sometimes limited to a certain part of the leg where it appeared as a bracelet.

Guinea pigs. Suspensions in a 1:50 dilution prepared from the infected brains of mice were injected into 10 guinea pigs intraperitoneally (1.0 cc.), into 10 guinea pigs subcutaneously (1.0 cc.), and into 10 guinea pigs intracerebrally (0.5 cc.).

The temperatures of the guinea pigs which had been injected intraperitoneally began to increase after 24 hours, two died after 48 hours, and three died during the fourth day. The temperatures of those injected subcutaneously increased after 48 hours and all died between the fourth and 10th days. A slight fever was observed in the guinea pigs which had received intracerebral inoculation and only two of them died after 12 days.

At autopsy, it was noted that all the guinea pigs had succumbed to hemorrhagic lobar pneumonia (fig. 13); no cerebral manifestations were observed in any of them.

Rabbits.

1. 0.1 cc. of the viral material cultivated in the allantoic fluid was inoculated into the vitreous body of five rabbits. No reaction was observed in two of them. In the remaining three, the vitreous became very cloudy. Small and large foci of chorioretinitis developed in various parts of the retina. The vitreous body of one rabbit was filled with exudate and a hypopyon appeared now and then in the anterior chamber.

2. 0.1 cc. of the 1:100 suspension of the viral material cultivated in the brains of mice was injected into the vitreous body of five rabbits. In all of them the vitreous became cloudy and various small and large foci of chorioretinitis were formed in the retina.

After 15 days the inside of the eye of one of them was filled with exudate and a thin hypopyon appeared. The hypopyon crisis recurred three times and the animal died after three months from encephalitis. The vitreous of the remaining four became cloudy from time to time, and there was pigment proliferation surrounding the lesions in the fundus.

3. 0.1 cc. of the 1:100 suspension of chorioallantoic membrane was injected into the vitreous body of five rabbits.

After two days, one of the animals showed a large mass in the inferior nasal quadrant of the fundus. The principal lesion was surrounded by tiny, very thin white spots.

Fig. 12 (Sezer). Disappearance of the hair of the mouse.

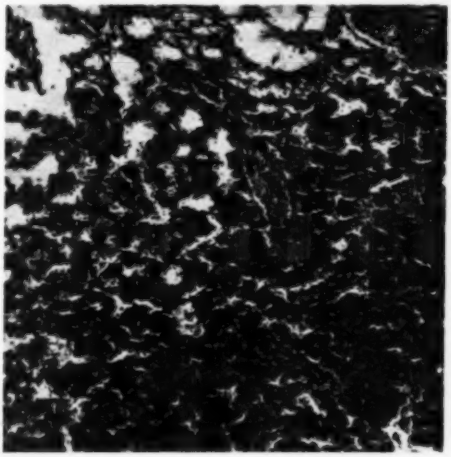

Fig. 13 (Sezer). Hemorrhagic lobar pneumonia of the guinea pig.

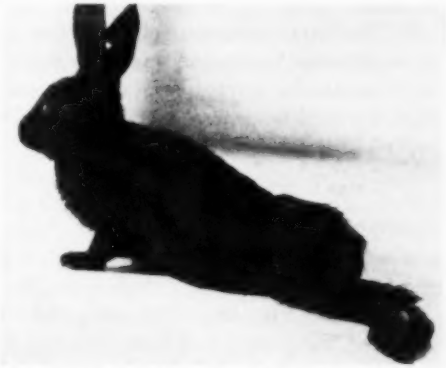

Fig. 14 (Sezer). Paralysis in the hind legs of the rabbit.

Fig. 15 (Sezer). Histopathologic signs in the brain of the rabbit (glial nodule).

Paralysis developed (fig. 14) in the hind legs after six days and the rabbit died on the eighth day. In the histopathologic examination of the brain, glial proliferation and typical nodular foci were seen (fig. 15).

In the remaining four rabbits, the vitreous became cloudy and small and large foci of chorioretinitis developed. The hair on the backs of three of the rabbits fell off but later reappeared (fig. 16). All four died from paralysis after 45 days.

4. 0.1 cc. of the suspension prepared from the spinal cord of the first rabbit in Experiment 3 which died (already described), was injected into the vitreous body of five rabbits. Foci of chorioretinitis developed in all of them.

5. The viral material cultivated on the chorioallantoic membrane was inoculated by friction into the scarred corneas of five rabbits. No local or general reactions were observed.

Characterization of the virus

Complement-fixation tests

Complement-fixation tests were made with the sera of 12 patients, including the three from whose eyes the virus was isolated. Some of these cases had been followed for years in the Clinic of Ophthalmology of the University of Istanbul and some had only lately applied for treatment. All of them manifested the usual clinical symptoms of Behçet's disease. In one (E. Kivilcim) the disease had first appeared in 1925. Both optic nerves were atrophied and the eyes had been blind for 10 years; by that time all manifestations of the disease had disappeared and had not again recurred.

Another patient (E. Karadeniz) had been ill since 1928. The optic nerves in both eyes were atrophied and the patient was totally blind. There had been no recent attacks of uveitis but there were still recurrent crises of aphthae on the scrotum and the mucosa of the mouth.

For the complement-fixation tests the following materials were used:

1. As antigens the allantoic fluid and mouse brain suspensions of the virus isolated

Fig. 16 (Sezer). Disappearance of the hair of the rabbit.

from three cases of Behçet's disease were used, and as control antigens normal allantoic fluid and normal mouse-brain suspensions were used. The complement-fixation tests were performed with the serum of each patient (or rabbit), and eight antigens, six of them specific and two normal. The tests were repeated after 10-day intervals.

2. The following 34 sera were used:

a. The sera of 12 patients with Behçet's disease.

b. The sera of three rabbits with experimental Behçet's disease.

c. The sera of five patients with iridocyclitis of a different etiology, and the sera of five patients with congestive glaucoma.

d. The sera of six patients whose eyes were not diseased, three of them having a +4 Wassermann reaction and the other three a negative Wassermann reaction.

e. The sera of three normal rabbits.

Results

a. Fixation was at the 1:128 level in five of the patients with Behçet's disease, at the 1:64 level in four, and at the 1:16 level in two. The result was negative in the patient (E. Kivilcim) who had shown no manifestations of the disease for 10 years.

b. Fixation was at the 1:64 level with the sera of the three rabbits with the experimental disease.

c, d, and e gave negative results.

The control tests made with normal allantoic fluid and the antigen of normal mouse brain also gave consistently negative results.

NEUTRALIZATION TESTS

The infected chorioallantoic membrane material was diluted in tyrode solution 1:10 and suspensions were prepared in the following concentrations: 1:10, 1:100, 1:1,000, 1:10,000, and 1:100,000. After being mixed in test tubes in equal amounts with the patient's serum which had been inactivated for half an hour at 56°C., the suspensions were left in the incubator for one hour at 37°C., and later in the ice box at 4°C. From each suspension, 0.1 cc. was inoculated into six chorioallantoic membranes. In a control series, normal human serum was used instead of the serum of a patient with Behçet's disease.

TABLE 1

NEUTRALIZATION OF THE VIRUS ON THE CHORIOALLANTOIC MEMBRANE WITH THE SERA OF PATIENTS AND NORMALS

Serum	Dilutions of the Virus				
	1:10	1:100	1:1,000	1:10,000	1:100,000
Normal	++++++	++++++	++++++	++++++	++++−−
Patient	++++++	+++++±	+++++±	±±−−−−	−−−−−−

Key: + = clear lesions on the chorioallantoic membrane.
± = doubtful or slight lesions on the chorioallantoic membrane.
− = normal chorioallantoic membrane.

FILTRATION OF THE VIRUS

Material from the 19th chorioallantoic-membrane passage of the first case was ground with sand, diluted in tyrode solution 1:10, and centrifuged at 2,000 r.p.m. A supernatant fluid was obtained. Half of this fluid was filtered through a Seitz filter with double pads.

The filtered and nonfiltered materials were inoculated in 0.25-cc. amounts into 12 chorioallantoic membranes. After 48 hours, half the embryos which were inoculated with either filtered or nonfiltered material were dead, and in the remaining six typical lesions had developed.

The nonfiltered material produced large lesions; the filtered material produced small and scattered lesions.

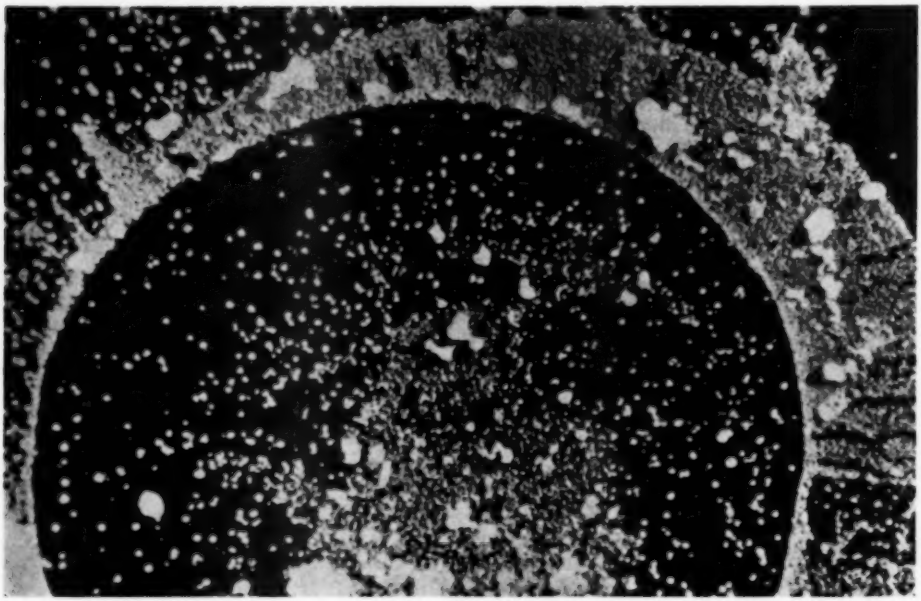

Fig. 17 (Sezer). Photograph of the virus taken with an electron microscope. A compact mass of elementary bodies.

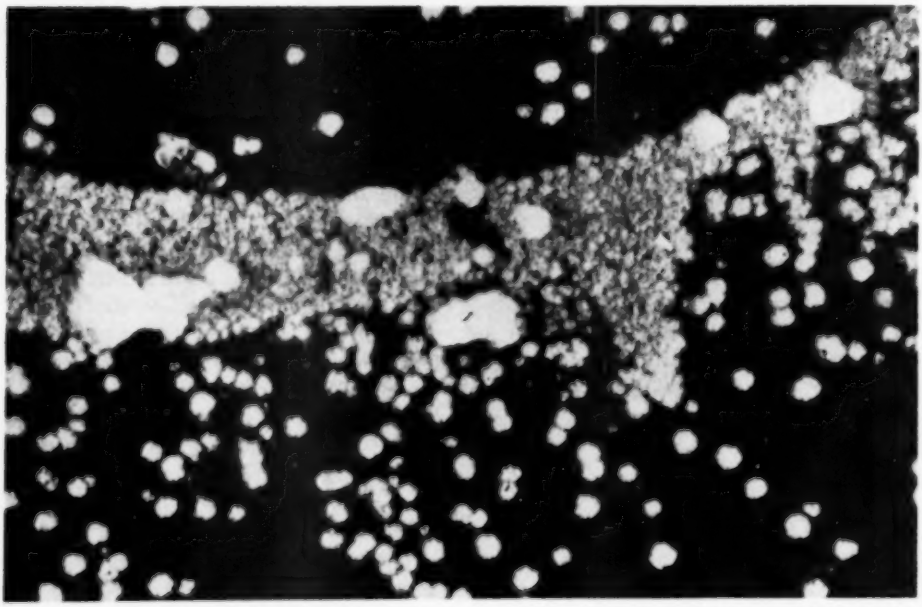

Fig. 18 (Sezer). Photograph taken with an electron microscope, showing a higher magnification of a section of Figure 17. The relation of the free bodies to the compact mass is clear.

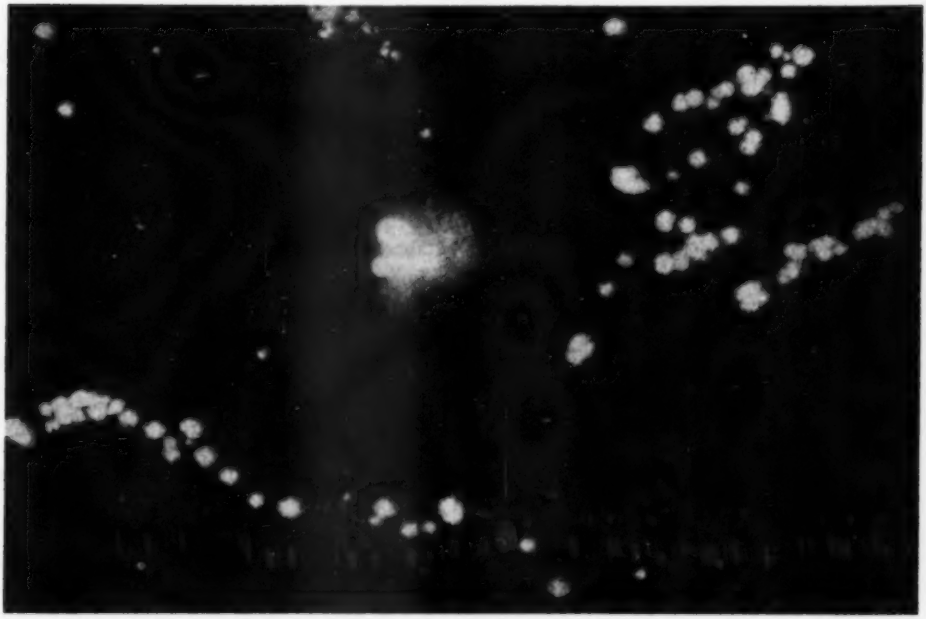

Fig. 19 (Sezer). Photograph of the virus taken with an electron microscope, showing a few of the more isolated bodies. The magnification is between those of Figures 17 and 18.

Relation of the virus to other viruses

On the basis of the results obtained from the serologic tests and experiments on animals, it was concluded that the pathologic agent discovered bears no relation to the viruses of herpes, lymphocytic choriomeningitis, or Theiler's virus.

A suspension of the virus in allantoic fluid was lyophilized and, through the courtesy of Dr. J. Stanley White of London Laboratories, was sent to Dr. Fred Stimpert at the Virus Research Laboratory of Parke, Davis, and Company, Detroit, Michigan, for examination with the electron microscope. In this laboratory, Dr. A. R. Taylor and Dr. I. W. McLean took electron microscope photographs of the virus which revealed the following:

Figure 17 shows a compact mass of elementary bodies and elementary particles surrounded by a ring of densely compacted particles. This ring formation may be a cellular remnant but is more likely the result of a group of desiccated elementary bodies around a micro air bubble. In Figure 18, which is a higher magnification of a section of Figure 17, the relation of the free bodies to the compact mass is clear. Figure 19 is a photograph of a separate field showing a few of the more isolated bodies; its magnification lies between those of Figures 17 and 18.

According to these photographs, the diameter of the virus particles is about 100 millimicrons (figs. 17, 18 and 19).

Discussion

In 1937 Behçet suggested that this syndrome, characterized by three cardinal symptoms, was an independent, morbid entity, caused by a specific agent, very probably a virus. Later, various writers accepted Behçet's hypothesis while others continued to be skeptical.

Behçet and the others who believed in a viral etiology were unsuccessful in their attempts to isolate the pathologic agent and

thus were obliged themselves to remain speculative. The cause of their failure appears now to have been the limitation of their investigations to the fluid in the anterior chamber and the exudate of the aphthae formed on the genitalia and in the mouth.

Our studies within these limits were equally unsuccessful. Only when our investigations were extended to the exudate collected from under the retina and from the vitreous body was it possible to isolate a virus. According to these findings, the pathologic agent of the disease apparently resides in the posterior segment of the eye and is probably absent from the mouth and genital organs as well as from the hypopyon.

The isolation of the pathologic agent from each of the three patients by the same method, and the development of exactly the same cultural and serologic properties in all three instances, would certainly seem to indicate that the disease in all three cases was caused by a common virus.

A strongly positive reaction obtained with this virus in complement-fixation and neutralization tests with the blood sera of 12 patients with the disease, and the absence of any reactions with normal human or animal sera, is considered corroborative evidence that the antigen discovered is the specific agent of this disease.

The virus caused manifestations in the eyes of rabbits almost like those in man and, in some cases, resulted in encephalitis and in cutaneous symptoms. It was also of interest in the experiments on mice that, in addition to encephalitis, cutaneous manifestations and signs of thrombophlebitis in the legs were of the same nature as those observed in some human patients.

Our studies would thus seem to confirm the supposition that this syndrome, with its three cardinal symptoms, is indeed an independent disease; and to establish its causative agent as a filtrable, neurotropic virus, with specific characteristics, first isolated in the course of these studies.

Conclusions

1. A virus was isolated from each of three patients with Behçet's disease. In each instance, it displayed the same cultural and serologic properties.

2. The virus caused typical encephalitis in mice and produced in the eyes of rabbits an experimental disease similar to the disease in man.

3. Positive complement-fixation and neutralization tests indicated that special antibodies against this virus are being produced in the blood of patients with the disease.

4. The virus multiplied in the chorioallantoic membrane of a fertile egg, in allantoic fluid, and in yolk sac, and caused the death of the embryo in a high percentage of experiments.

5. The virus killed guinea pigs by causing hemorrhagic lobar pneumonia.

6. The virus passed through a Seitz filter with double pads.

7. According to electron microscope photographs, the diameter of the virus particles is about 100 millimicrons.

Acknowledgments

I should like to thank Dr. Mes'ut Okan for her valuable technical assistance; Prof. Ihsan Sukru Aksel, director of the Department of Neurology, University of Istanbul, Prof. Hogan of the University of California, and Dr. Bedrettin Pars, pathologist of the Cerrahpasa Hospital, Istanbul, for their kind help in histopathologic examinations; Dr. J. Stanley White of London Laboratories, Dr. Fred Stimpert, Dr. A. R. Taylor, and Dr. I. W. McLean for making the examinations with the electron microscope and for taking the photographs. I also appreciate the kind help of Prof. Naci Bengisu, director of the Department of Ophthalmology, University of Istanbul, in the clinical control of the cases.

References

1. Behçet, H.: Ueber rezidivierende, aphthöse, durch ein Virus verursachte Geschwüre am Mund, am Auge und an den Genitalien. Dermat. Wchnschr. **105**:115, 1937.

2. ———: Consideration sur les lesions aphteuses de la bouche et des parties génitales ainsi que sur les manifestations acolaires d'origine probablement parasitaire et observations concernant leur foyer d'infection. Bull. Soc. franç. de dermat. et syph., **43**:420, 1938.

Apropos d'une entité morbide, due probablement à un virus spécial donnant lieu à une infection généralisée, se manifestant par des poussées récidivantes en trois régions principales et occasionnant en particulier des iritis répétées. Bull. Soc. franç. de dermat. et syph., **46**:674, 1939.

Some observations on the clinical picture of the so called triple symptom complex. Dermatologica, **81**:73, 1940.

3. Weekers, L., and Reginster, H.: Un nouveau syndrome: iritis, ulcères aigus de la bouche et de la vulve. Sa parenté avec l'iritis récidivante à hypopyon. Arch. d'ophtal., **2**:697, 1938.

4. Franceschetti, A.: Les affection oculaires d'origine rhumatismale. Ophthalmologica, **111**:242, 1946.

Franceschetti, A., Valerio, M., and Babel, J.: Recurrent aphthous uveitis with mucocutaneous lesions. Arch. Ophth., **35**:469, 1946.

5. Wise and Sulzberger: Yearbook of Dermatology and Syphilis. Chicago, Yearbook, 1940, p. 35.

6. Jensen, T.: Recurrent aphthous ulcerations of oral mucous membrane and genitals associated with recurrent hypopyon, iritis, and atrophy of optic nerve. Ugesk. f. laeger., **102**:1023, 1940.

Sur les ulcérations aphteuses de la muquese de la bouche et de la peau génitale combinées avec les symptoms oculaires. Acta dermat. venereol. **22**:64, 1941.

7. Mach, R., Babel, J., and Naville, N.: Syndromes muco-cutanés avec complications oculaires (érythème polymorphe iritis récidivante aphteuse), Helvet. med. acta., **7**:552, 1941.

8. Meyer, G.: Soc. Ophth. Utrecht, 1942, p. 14.

9. Babel, J.: Iritis et iridocyclites. Ophthalmologica, **105**:318, 1943.

10. Jacobini: Proc. A. Am. Physicians, 1894, p. 279.

11. Sutton: From Behçet's bibliography.

12. Neuschuler: Negels Jahresbuch, 1898.

13. Reis: Klin. Monatsbl. f. Augenh., **44**:203, 1906.

14. Blüthe: From Behçet's bibliography.

15. Koppe: Arch. f. Ophth., **85**:94, 1913.

16. Gilbert: Arch. f. Augenh., **87**:27, 1921.

17. Weve: Arch. f. Augenh. **93**:14, 1923.

18. Fuchs, A.: Ueber chronische multiple knotenbilding am Körper mit haufig rezidivie render eitriger Iritis und Skleritis. Deutsche Med., **36**:1053, 1926.

19. Carol: Med. Tijdschr., **1**:369, 1926.

20. Adamantiades, B.: La trombophlébite comme quatrième symptome de l'iritis récidivant à hypopyon. Ann. d'ocul., **179**:143, 1946.

Sur un nouveau cas de syndrome complexe d'iritis récidivante à hypopyon. (Reference in Ann. d'ocul., **183**:628, 1950).

Recurrent uveitis. Am. J. Ophth., **34**:447, 1951.

Adamantiades, B., and Lorando, N.: Sur le syndrome complexe de l'uvéite récidivante ou soi-disant syndrome complexe de Behçet. Presse méd., **57**:501, 1949.

21. Cavara, V.: Nouve osservazioni ed ulteriori ricerche sull'ipopionirite recidivante associata ad alterazioni delle mucose e della cute. Bol. d'ocul., **29**:789, 1940.

Cavara, V., and Bietti, G. B.: Le Manifestazioni Oculari Delle Malattie da Virus e da Rickettsie, 1952.

22. Withwell: Brit. J. Dermat., **46**:414, 1934.

23. Urbanek: Ztschr. f. Augenh., **83**:357, 1934.

24. Alm, L., and Obery, L.: Nord. Med., **25**:603, 1945.

OCCLUSION OF THE CENTRAL RETINAL VEIN*

Clinical importance of certain histopathologic observations

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Most investigators of the mechanism of occlusion of the central retinal vein agree that simple primary thrombosis in this vessel, if it occurs at all, must be extremely rare. Clinical evidence suggests its existence in certain blood dyscrasias with pronounced thrombotic tendencies (Jürgens and Bach,[1] Rowlands and Vaizey,[2] Klien[3]), in trauma, and in septic conditions.

Usually the occlusion is the result of varying combinations of three factors, namely:

1. Sclerotic and degenerative changes in the venous wall itself.

2. Sclerotic and degenerative alterations of the adjacent structures, namely the central artery, the central connective-tissue strand, and the cribrous plate, complicated in some cases by preëxisting unfavorable anatomic relations.

3. Stasis producing disturbances of the hemodynamics of the retinal circulation, such as occur, for instance, in certain phases of systemic hypertension or in marasmus.

Almost all investigators of the problem have found a prevalence of intimal changes in the vein, although their interpretation of the phenomenon varies.

Pathologic studies of eyes lost after occlusion of the central vein reveal the surprising fact that these endothelial lesions are very similar regardless of whether the occlusion occurred in an aged individual with simple senile angiosclerosis or arteriosclerotic hypertension or in a younger patient with diabetes or essential hypertension of long duration. They consist of cushionlike proliferations of the endothelium in the intralaminar portion of the vein and, in some cases, of extensive detachments of the endothelial lining forming dissecting aneurysms.

These almost standard intimal lesions may be combined with prominent sclerotic findings in the remaining constituents of the venous wall and in the central artery and the surrounding tissues. Led by these observations Harms,[4] and later Scheerer[5] and Hertel[6] considered the endothelial proliferation secondary to a compression of the vein from the outside and intimal irritation after partial collapse of the vein. Verhoeff,[7] on the other hand, favored the conception of a primary endophlebitis and degeneration of the endothelium. He showed that the intimal lining may be forced away from the venous wall by the blood stream of tributary veins in the manner of a dissecting aneurysm. Subdivision of the venous lumen by such endothelial proliferations was also interpreted as recanalization of a thrombus (Coats,[8] Sidler-Huguenin[9]).

In a certain percentage of cases the occlusive mechanism is demonstrable as a direct complication of the intimal damage, as has been discussed elsewhere (Klien[3, 10]), and as is shown herein in Case 1.

More often the immediate cause of the occlusion is not apparent histologically, suggesting a combination of endothelial degeneration with stasis producing disturbances of the hemodynamic mechanism of the retinal circulation. In this connection the vicious circle which may be initiated by irritation of the inner surface of a venous wall, and which consists of a reflex constriction of the arteries in the corresponding area (Leriche[11]) should not be underestimated.

Small knobs of endothelial proliferation or a flapping motion of a detached sheath of endothelium within the central vein could produce a reflex spasm of the central artery whose walls lie so close to it, and of its retinal branches, thus slowing down further the circulation in the venous retinal tree.

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From the histologic appearance of a fibrous plug in the venous lumen which is several months old, it often can no longer be determined how much of it was primary endothelial proliferation and how much the product of organization by ingrowth of subendothelial connective tissue of a secondary thrombus. If the clinical and histologic analysis of a case reveals little anatomically demonstrable angiosclerosis and much evidence of hemodynamic disturbances prior to the venous occlusion, then analogies with pathologic anatomic processes elsewhere in the body make at least probable, a terminal thrombotic factor in the occlusive mechanism.

The two following cases illustrate two different and somewhat unusual principal factors in the mechanism of venous occlusion. The first case is an intramural thrombus arising from degeneration and detachment of the intimal lining; the second case is a marked degree of primary phlebosclerosis affecting the fibrous part of the venous wall.

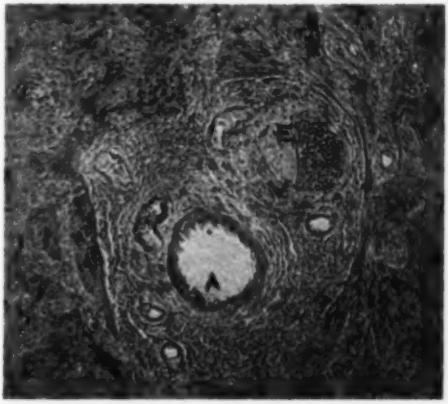

Fig. 1 (Klien). *Case I.* Cross sections of central vessels at level of the posterior scleral surface. A = artery, V = vein. The venous endothelium (E) is detached by a serous coagulum. Slightly more than half of the lumen is well filled with blood.

CASE 1

History. L. M., a woman, 58 years of age, had suffered from hypertensive cardiovascular disease for several years but was asymptomatic most of the time. Her systolic blood pressure varied from 180 to 200 mm. Hg; the diastolic pressure was 90 mm. Hg. During the period of hospitalization for removal of the right eye, her blood pressure was 150/90 mm. Hg and there was imminent cardiac failure.

Four and a half months prior to the removal of her right eye she had noted one afternoon, while doing her laundry work, a gradually increasing haziness of her right vision. Within two days all vision in this eye with the exception of the extreme temporal field was lost, and after several more days even this visual remnant disappeared.

One month after this incident the right eye became painful and, excepting a short period of remission, remained so up to the time of enucleation.

Findings. Vision of the right eye was nil; left eye, normal. The cornea of the right eye was edematous and prevented a view of the deeper structures. The intraocular pressure in the right eye was markedly increased.

The left eye was externally normal. In the left fundus the optic disc was normal. There was moderate tortuosity and some sclerosis of the retinal arteries, but no attenuation of the arterial tree, no arteriovenous compression or retinopathy.

Histologic findings. The globe was fixed in Mueller's solution. Serial cross sections of the entire segment of the optic nerve attached to the eyeball (1.5 mm.) and of its intraocular portion were made.

One millimeter behind the cribrous plate the central artery and vein were still separate, each being carried by its own connective-tissue strand. At this retrolaminar level there was a slight concentric narrowing of the venous aperture caused by moderate uniform thickening of the adventitia. There was also moderate thickening of the perivascular sheath of the central artery, but not of its muscularis or intima.

At the level of section 111 (fig. 1), still behind the cribrous plate, the central artery and vein were already carried by the common central connective-tissue strand. The endothelium of the vein bulged toward the

lumen in one area, owing to edematous imbibition of the venous wall in this region.

In section 121 (fig. 2), still in the retrolaminar portion of the nerve, the blood from a tributary venous branch was seen to have forced the endothelium far into the lumen. The detached endothelium appeared wrinkled and rolled up in the center, suggesting a break in its continuity near this level.

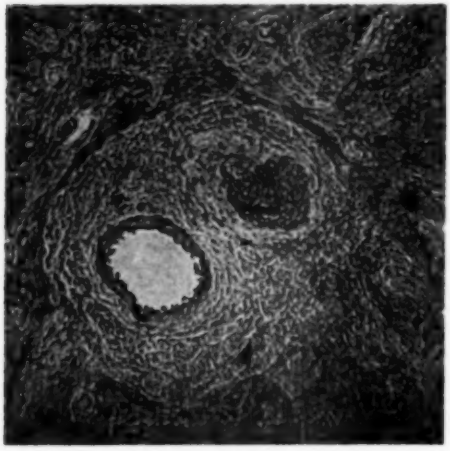

Fig. 2 (Klien). *Case 1.* Cross section of the central vessels, still at a level behind the lamina cribrosa. The endothelium of the central vein is forced into the lumen by blood from a small tributary branch.

In section 127 (fig. 3) an intralaminar location near the posterior surface of the cribrous plate had been reached. It showed the venous lumen reduced to a small oval area by an extensive detachment of the endothelium. The space between the detached endothelium and the venous wall was filled with spider-weblike formations of proliferated endothelium between a serous coagulum containing a few red blood cells.

Section 136 (fig. 4) illustrates the intralaminar appearance of the central vessels within the central portion of the cribrous plate. Neither artery nor vein had uncommonly thickened walls at this level. The vein was entirely uncrowded by the adjacent structures in spite of a slight thickening of the central connective-tissue strand, but its lumen was completely obstructed by an intramural (subendothelial), almost structureless thrombus containing only a few proliferated endothelial cells and subendothelial fibroblasts.

From this level distalward there was in-

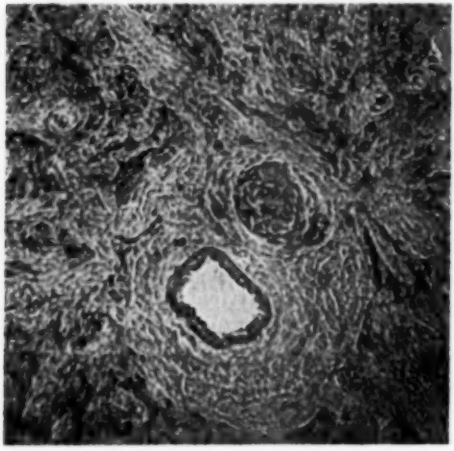

Fig. 3 (Klien). *Case 1.* The central vessels within the lamina cribrosa near its posterior surface. The venous lumen is reduced to a slit by extensive detachment of the endothelium. The subendothelial space is filled with a reticulum of proliferated endothelial cells and fibroblasts, whose meshes contain a serous coagulum and a few erythrocytes.

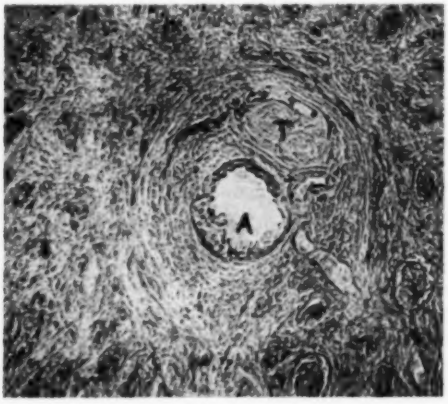

Fig. 4 (Klien). *Case 1.* The central vessels near the center of the cribrous plate. The central connective tissue strand is moderately thickened, but artery and vein appear uncrowded. The lumen of the vein is reduced to a slit by an almost structureless intramural thrombus (T) which is continuous with the subendothelial coagulum shown in Figure 3.

creasing organization and degeneration of the intramural thrombus. Section 139 revealed the hyalin degeneration (van Gieson stain) of portions of the thrombus.

From the center of the cribrous plate through its anterior portion the venous aperture became increasingly wider. In section 140 (fig. 5) a small, well-defined atheroma of the central artery was encountered but it did not cause a compression of the vein.

At the level of the junction of the superior and inferior retinal veins, with a smaller third branch joining at the same place (section 147, fig. 6), the venous lumina were wide and the most anterior tip of the intramural thrombus was discovered in the wall of one of the main branches.

Description of the meridional sections of the globe. There was mild pigment dispersion over the posterior surface of the cornea. The angle of the anterior chamber was blocked almost in its entire circumference by anterior synechias of varying extent. The anterior surface of the atrophic iris was partly covered by neovascularization.

There was widespread hemorrhagic imbibition of the retina and some free hemorrhage in the posterior vitreous. The latter was partly organized into a thin fibrous membrane which covered the inner surface of the retina and optic nervehead. The prelaminar portion of the optic nerve was completely atrophic and contained numerous newly formed blood vessels. Also the inner layers of the retina contained small convolutions of new vessels. There was a marked degree of retinal arteriolar sclerosis.

The choroidal arteries also had thick fibrous and partly hyalinized walls. In several places there were convolutions of new capillaries and solid heaps of proliferated endothelial cells surrounded by dense foci of round-cell infiltration.

Comment

The occlusion of the central vein in this case was caused by an intramural thrombus, which had its inception in the retrolaminar region, where blood from a tributary had been forced behind the ruptured and detached intimal lining. This thrombus could be followed through the entire intralaminar portion of the vein forward into the wall of one of its main branches.

The site of the complete occlusion of the venous aperture was intralaminar near the posterior surface of the cribrous plate. Pos-

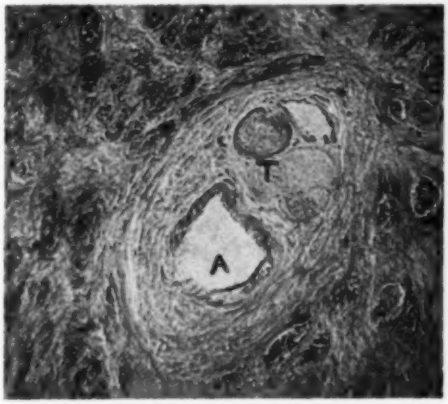

Fig. 5 (Klien). *Case 1.* Cross section of the central vessels in anterior half of the lamina cribrosa. Van Gieson stain reveals hyalin degeneration of the intramural thrombus. Venous lumen wider, degenerated thrombotic mass (T) between adventitia and endothelium of vein. Small atheroma of central artery.

Fig. 6 (Klien). *Case 1.* Near anterior surface of cribrous plate. Junction of the superior and inferior retinal veins and a third smaller branch. The most anterior hyalin tip of the intramural thrombus (T) is visible in the wall of one of the major venous branches.

terior to this place there appeared to be a break in the intimal lining as suggested by the rolled edge of the detached endothelial sheath.

Nowhere was the vein compressed by its neighboring structures, a fact which points to a primary disease of the vein, in this case affecting mainly the endothelial lining.

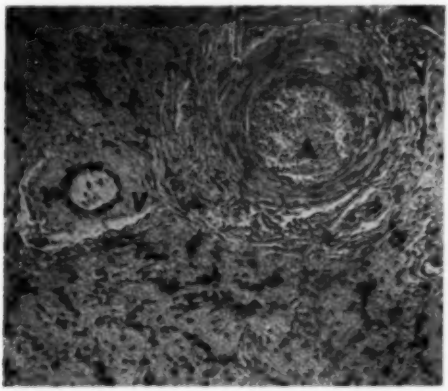

Fig. 7 (Klien). *Case 2.* Cross sections of central vessels one mm. behind globe. A = Artery, V = vein. Marked thickening and hyalin degeneration of venous wall, narrow lumen even at this level. Puckering of endothelium at one place.

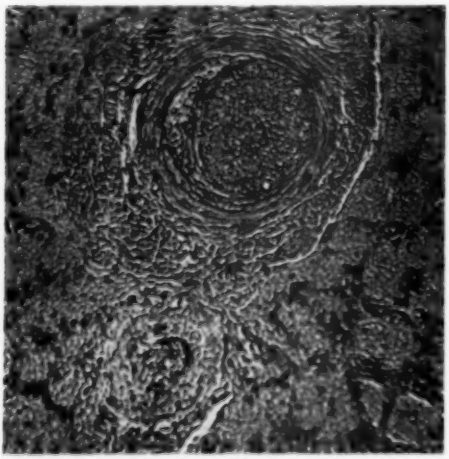

Fig. 8 (Klien). *Case 2.* Cross section of central vessels at a level just behind the posterior surface of the cribrous plate. The central connective-tissue strand and the walls of the central artery are not thickened. There is a subendothelial atheroma in the central artery. Isolated phlebosclerosis. Hyalin degeneration of thickened venous wall, indicated by Van Gieson stain.

The clinical history of this patient points to an initial partial occlusion which became complete within four days. It is impossible to draw definite conclusions as to whether the time of the incipient visual disturbance was coincident with extensive tearing of the endothelium which could have caused a relatively rapid occlusion and could have initiated the intramural thrombus formation at the same time, or whether this process had been under way for a longer period before reaching the occlusive stage, the tear being incidental to organization and longitudinal shrinking of the thrombus.

Two facts seem to favor the latter conception: first, the length of the intramural thrombus, which suggests a low-grade force over an extended period of time rather than a sudden event; second, the severe degree of retinal angiosclerosis in this eye, while only the mildest form of it was observed in the fellow eye. Three months and a half of varying degrees of increased intraocular pressure would not seem to be sufficient for production of such a degree of unilateral angiosclerosis.

Observations of the influence upon the arterial retinal tree of intermittent or repeated partial venous occlusions, which give rise to unilateral angiosclerosis, suggest also for this case a possibility of chronic intimal irritation in the vein with repeated reflex spasms in the retinal arteries, resulting in pronounced localized sclerosis.

It would have been interesting to know if unilateral retinal angiosclerosis or a more pronounced degree of it existed in this eye prior to the occlusion. Such ophthalmoscopic findings in hypertensive fundi should be noted. In their presence prophylactic treatment with anticoagulants combined with antispasmodics might avert, delay, or favorably modify the visual catastrophy.

Case 2

History. C. S., a woman, 78 years of age,

was suffering from severe systemic hypertension with mild cardiac decompensation at the time of the onset of the ocular complaints.

Failing vision in the right eye had been noted for the past four months and pain in this eye began three weeks prior to its removal.

Findings. Vision in the right eye was nil; the left eye, normal. The cornea of the right eye was steamy, the anterior chamber shallow, the pupil irregular and dilated. The tension was 75 mm. Hg (Schiøtz). The intraocular pressure of the left eye was normal.

Histologic findings. The right globe was fixed in formalin. Serial cross sections of the entire portion of optic nerve attached to the globe (1.0 mm.) and of its intraocular portion were made.

There was pronounced thickening and hyalin degeneration of the adventitia of the central vein in its retrolaminar portion, causing concentric narrowing of the lumen and in several places puckering of the endothelial lining, probably due to contraction of the fibrous tissue (section 160, fig. 7 and section 181, fig. 8). At this level the tubular lumen contained a small amount of blood.

In section 201, just behind the cribrous plate, the vesicular degeneration of the endothelium, so prominent in the more distal sections, was beginning to appear, reducing the lumen to a slit.

The tremendous thickening of the venous wall ceased abruptly at the posterior surface of the lamina cribrosa and throughout the intralaminar course the adventitia had normal dimensions.

Within the posterior half of the cribrous plate the lumen of the vein was completely occluded by swollen, degenerating cells, apparently endothelial cells which were still partly in the continuity of the intimal tube, partly desquamated and mixed with a few proliferated subendothelial fibroblasts.

In section 215 (fig. 9), at a level immediately behind the junction of the superior and inferior retinal veins, the lumen was still occluded mainly by these cells, but a few polymorphonuclear leukocytes and small amounts of plasma or fibrin were beginning to be wedged in between them.

Just prior to the junction (section 217, fig. 10) the lumina of the two veins were filled with plasma and leukocytes with a few hydropic intimal cells between them.

In the prelaminar region the two veins were slightly distended and well filled with blood which still contained a large number

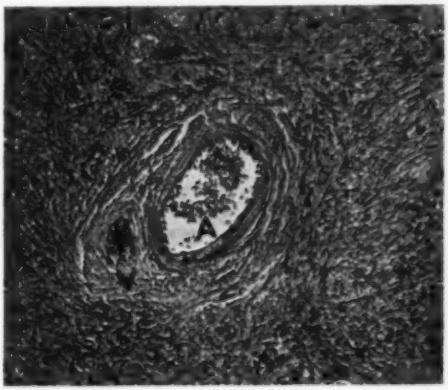

Fig. 9 (Klien). *Case 2.* Cross section of central vessels in center of lamina cribrosa, immediately after junction of superior and inferior retinal veins. Narrow lumen of vein filled with hydropic endothelial cells and a few leukocytes wedged in between them.

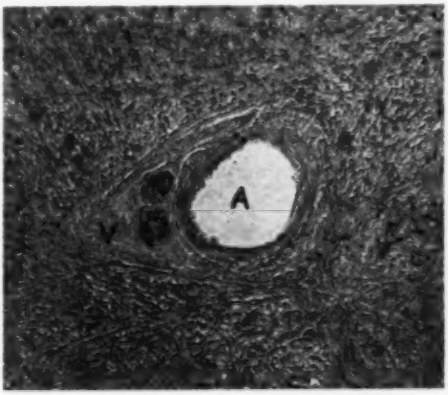

Fig. 10 (Klien). *Case 2.* Superior and inferior retinal veins just before junction. Their lumina are filled with plasma and leukocytes. A few hydropic endothelial cells are still visible.

of white cells. A small tributary venous branch was found occluded by hydropic swelling of its endothelial cells similar to that encountered in the main stem (fig. 11).

The structures contiguous with the central vein were remarkably free of hypertrophy and degeneration. The central connective-tissue strand had entirely normal dimensions and constituents. There was only mild thickening of the adventitia of the central artery with one small atheroma in its retrolaminar course which gave rise to a circumscribed intimal irregularity in the arterial lumen without confining the space for the vein.

Fig. 11 (Klien). *Case 2.* A small tributary venous branch in the prelaminar region is occluded by hydropic swelling of its endothelial lining (H), similar to that of central vein, shown in Figure 9.

Description of the meridional sections. The angle of the anterior chamber was obliterated by short anterior synechias and newly formed capillaries, which had begun to form a vascularized membrane on the anterior surface of the iris.

There was diffuse hemorrhagic imbibition of the retina and pronounced retinal arteriolar sclerosis mainly of the hyalin type. Small convolutions of new capillaries were embedded in the inner layers of the retina and within the remnants of the nervehead left in the meridional sections.

COMMENT

The occlusion of the vein occurred within

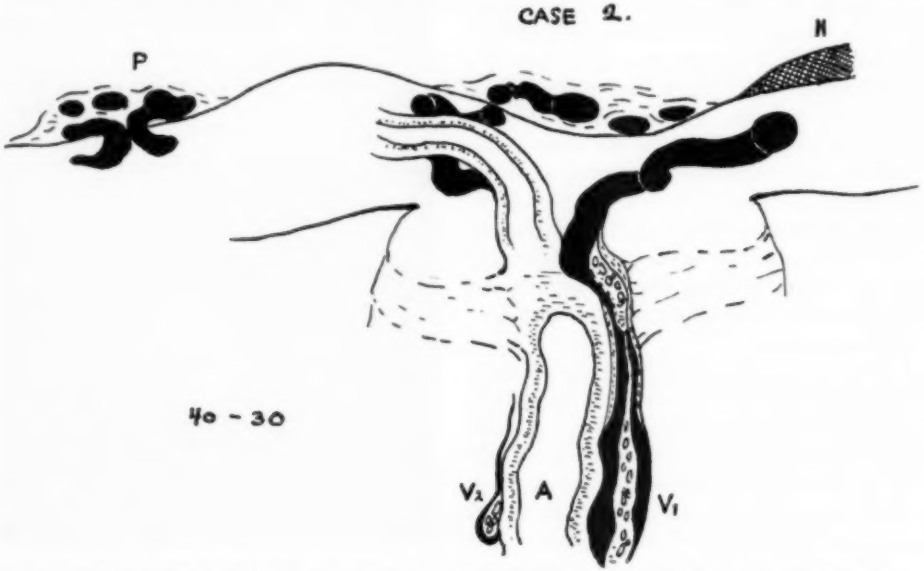

Fig. 12 (Klien). Reproduction of illustration from the AMERICAN JOURNAL OF OPHTHALMOLOGY, **27**:1343, 1944 (Klien). Vascular profile of central vessels reconstructed from the eye of a patient with severe diabetes. Note the cushionlike proliferation of the venous endothelium within the intralaminar course and its extensive detachment further back.

the posterior and middle third of the lamina cribrosa and appeared to be due to hydrops and desquamation of intimal cells in an otherwise uncrowded vessel. The endothelial degeneration in this case seemed different in character from that in the first case, mainly in that it seemed not to be part of the primary venous disease but secondary to local conditions.

One might conceive of the pathogenesis of occlusion in this case as a very slowly developing embarrassment of the venous flow by concentric narrowing of the retrolaminar lumen of the vein caused by an unusual degree of primary phlebosclerosis. In this aged patient with systemic hypertension and incipient cardiac decompensation, hemodynamic disturbances may have added to impairment of the venous circulation.

With the increasing stasis and malnutrition of the vessel wall in the intra- and prelaminar sections of the vein, secondary intimal damage in the form of hydrops and desquamation of the endothelium also advanced, eventually occluding the lumen. The decomposition of the stagnant blood column into fibrin, plasma, and leukocytes found in the vein and its main tributaries distal to the occluded segment must be an entirely secondary phenomenon and cannot be interpreted as a primary thrombus.

Summary and Conclusions

1. Histopathologic analysis of two cases of occlusion of the central retinal vein revealed two different principal factors in the occlusive mechanism, not hitherto described; namely, primary endothelial degeneration with secondary intramural thrombus formation in one case, and extreme phlebosclerosis with secondary endothelial deterioration in the other. Both cases have in common an almost isolated primary disease of the vein without the degenerative or sclerotic alterations of the surrounding structures, which are so often companion lesions in occlusive disease of this vessel.

2. These two instances of venous occlusion developing in patients with systemic hypertension supplement a previous study by me (Klien[12]) of 17 cases, seven of which occurred in systemic hypertension, six in arteriosclerotic disease, and four in diabetes mellitus. In all of these eyes, vascular profiles were reconstructed as shown in the reproduction of one of them (fig. 12). Five of these revealed detachments of the endothelial lining extending backward well beyond the posterior surface of the cribrous plate similar to that in Case 1 herein reported. Cross sections of the central vessels in these cases might have resembled those shown in Figures 1 to 5.

3. The clinical implications of these studies are, on the one hand, a sound explanation of the general experience that treatment in the majority of cases of an already established occlusion is of little value excepting perhaps in the rare instances occurring in blood dyscrasias, sepsis, and trauma.

The rationale of prophylactic therapy, on the other hand, in the more common types of occlusive venous disease complicating systemic hypertension, arteriosclerosis, and diabetes, gains support. Prophylactic therapy in these cases has three objectives, as discussed in detail elsewhere (Klien[12]): (a) to permit repair of incipient damage to the venous endothelium by reducing stress upon the vessel wall; (b) to gain time for the development of patent collateral channels if the occlusion is eventually inevitable; and (c) to lessen the danger of secondary thrombus formation at the site of the damaged vessel wall, especially during periods of added venous stasis such as occur in a multitude of systemic conditions through a sudden reduction of blood pressure.

It should be emphasized in this connection that anticoagulant therapy, without or in combination with antispasmodics, in a patient with occlusion of the central retinal vein or of one of its branches, may be preventive in regard to the fellow eye. Even if the vision of the eye with the already accomplished occlusion does not improve signifi-

cantly, the treatment should not be considered useless. Detailed observation of the fellow eye often reveals improvement of its vision, or more dramatically, disappearance of some subjective and objective signs of incipient impairment of its venous circulation.

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References

1. Jürgens, R., and Bach, K.: Thrombosebereitschaft bei Polycythaemia vera. Deutsches Arch. f. klin. Med., **176**:626, 1934.
2. Rowlands, R., and Vaizey, J. M.: Primary thrombocythaemia. Lancet, **2**:1217, 1938.
3. Klien, B.: Anticoagulant therapy of occlusion of central vein of retina. Arch. Ophth., **29**:699, 1943.
4. Harms, C.: Anatomische Untersuchungen der Gefässerkrankungen im Gebiete der Arteria und Vena Centralis. Arch. f. Ophth., **61**:1, 1905.
5. Scheerer, R.: Zur pathologischen Anatomie der Netzhaut-Zentralgefässe. Arch. f. Ophth., **112**:218, 1923.
6. Hertel, O.: Beitrag zur Angiosclerose der Zentralgefässe des Auges. Arch. f. Ophth., **52**:191, 1901.
7. Verhoeff, F. H.: Obstruction of the central retinal vein. Arch. Ophth., **36**:1, 1907.
8. Coats, G.: Complete obstruction of the central vein of the retina. Royal London Ophth. Hosp. Rep., **16**:262, 1904.
9. Sidler-Huguenin: Abhebung des Endothelrohres in der Zentral Arterie und kanalisierter Thrombus in der Zentral Vene. Arch. f. Augen., **1**:27, 1904.
10. Klien, B.: Obstruction of the central retinal vein: A clinicopathologic analysis. Am. J. Ophth., **27**:1339, 1944.
11. Leriche, R.: Quoted by E. Hartmann: Arch. Ophth., **28**:605, 1942.
12. Klien, B.: Prevention of retinal venous occlusion with special reference to ambulatory dicumarol therapy. Am. J. Ophth., **33**:175, 1950.

RETINAL VASCULAR CHANGES IN DIABETES MELLITUS*

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Visual impairment has too frequently accompanied increased length of life in patients with diabetes mellitus. It has been found that the incidence has at least tripled since longevity has been made possible for these patients.[1, 2] Diabetic retinopathy has been established as a clinical entity since Leber's original description of the ophthalmoscopic picture. Since these retinal changes are frequently found in cases in which the range of blood pressure is normal and there is no arteriosclerosis, it would seem that they are not essential pathogenic factors.[2-4] It has been observed that the capillaries themselves must be involved as the ophthalmoscopically visible vessels are frequently without notable change.[5]

*From the Cincinnati General Hospital. Presented at the annual meeting of the East Central Section of the Association for Research in Ophthalmology, Ann Arbor, Michigan, February 1952.

Present Study

Twenty-one cases of diabetes mellitus and an equal number of cases in which this disease is not known furnish the flat retinal specimens for this investigation. The retinas were obtained at autopsy and were examined by one or more methods, including inspection of unstained, injected, and stained preparations. Filtered India ink was chosen as a convenient nondialyzable substance for injection into the retinal vessels. The technique for injecting cut posterior segments was facilitated by using the dural sheath as a funnel for placing a No. 27 cannula into the central retinal artery at its site of entrance. The Hotchkiss-McManus technique, as described by Friedenwald,[6] is used as another means of demonstrating the vascular network.

In this series, the changes in the larger

vessels were not marked, except in specimens from patients who had had arteriosclerosis or hyperpiesia. The group with diabetes mellitus and the control group were divided into those cases without arteriosclerosis or hyperpiesia, those with arteriosclerosis without an elevation of diastolic pressure over 95 mm. Hg and those cases with both arteriosclerosis and diastolic pressures over 95 mm. Hg. All of the cases with hyperpiesia were found to have arteriosclerosis either in the retinal or in other tissue examined at autopsy, or in both.

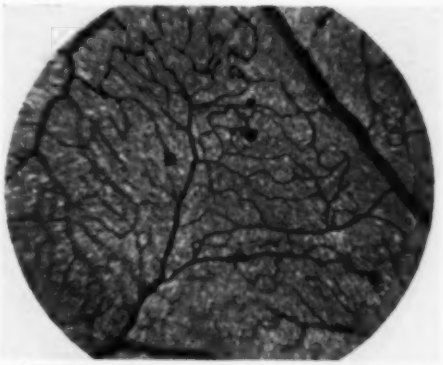

Fig. 1 (Hartford). An example of capillary network in which the position of the aneurysm has little preference for either the venular or the arteriolar side (×235).

Microaneurysms

Occurrence of microaneurysms without other changes in the fundus has been suggested as the earliest sign of diabetes mellitus[4] and, in this disease, the capillaries have been noted to show the greatest disintegration.[7]

In this series all of the diabetics without arteriosclerosis and hyperpiesia had microaneurysms; six of the eight diabetics with arteriosclerosis but no hyperpiesia had these changes; and, in the seven diabetic cases in which both arteriosclerosis and hyperpiesia were present, all had microaneurysms. These aneurysms occurred throughout the capillary network and were more numerous toward the venular side (figs. 1 to 6).

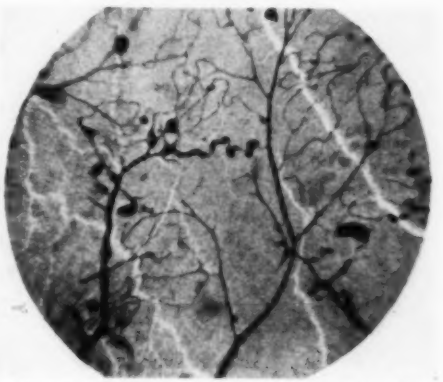

Fig. 2 (Hartford). Areas in which the microaneurysms are in the capillaries toward the venular side.

Supporting the endothelium of the capillary is a basement membrane containing a polysaccharide, which, after being oxidized to aldehyde by periodic acid, will stain with fuchsin sulfite. There are aneurysms in which the wall is not thicker than that of a normal capillary (fig. 7), and hyaline thickenings of the basement membrane can occur when there are no aneurysms.[1] Many of the microaneurysms reported herein were found to have markedly thickened walls which appeared to be continuous with the basement membrane (fig. 8).

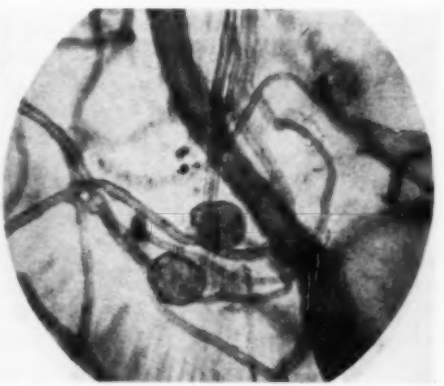

Fig. 3 (Hartford). Globular microaneurysms (×1035).

Some components of the material contributing to this thickening have been found to be mucoid or mucoprotein.[1] In these, the polysaccharide fraction is reported as

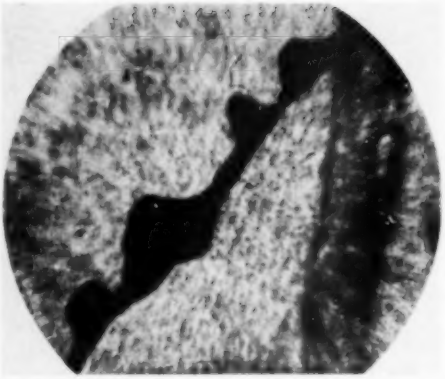

Fig. 4 (Hartford). Fusiform microaneurysms.

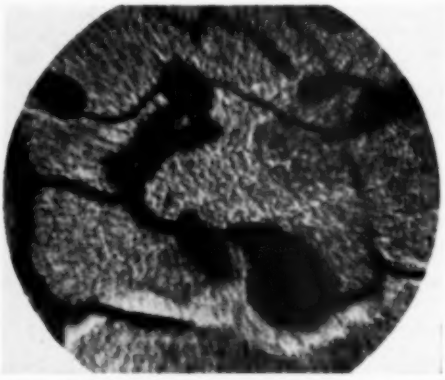

Fig. 5 (Hartford). Cylindrical forms, some of which fold upon themselves as though the contacting surfaces were merging.

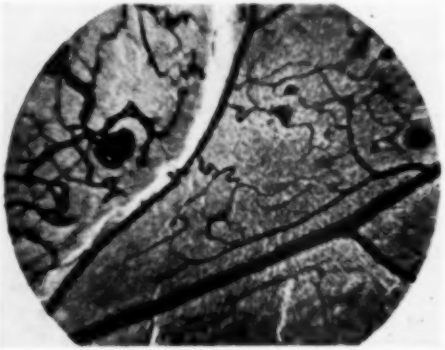

Fig. 6 (Hartford). Bizarre forms and capillary looping.

firmly bound to a protein component. The content of hexosamine is considerable.[8] Some studies of protein metabolism in clinical and in experimental alloxan diabetes suggest that derangement of protein metabolism may be the factor at fault.[2] Retinal lesions have been noted to develop with dramatic rapidity in rabbits deficient in plasma albumin when diabetes was superimposed by administration of alloxan.[9]

The composition of the thickenings of the basement membrane is not uniform. Some thickenings contain appreciable lipid ma-

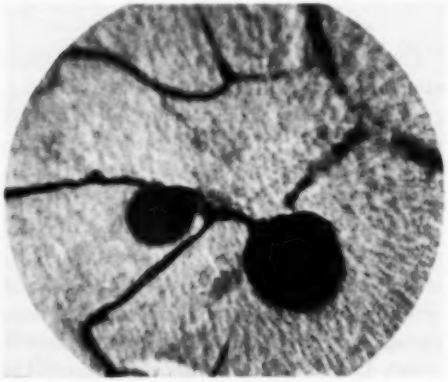

Fig. 7 (Hartford). No thickening of the basement membrane.

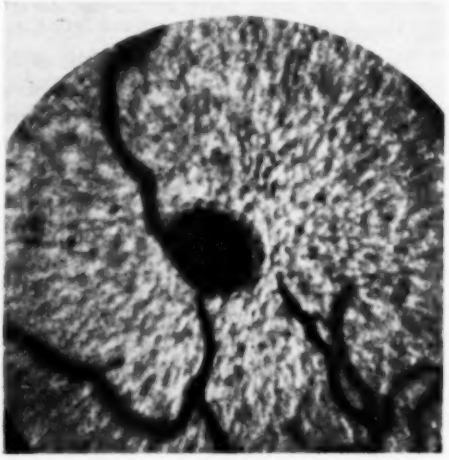

Fig. 8 (Hartford). Capillary aneurysm with thickening of the affected area of the basement membrane.

terial (fig. 9). Ballantyne and Loewenstein[10] have found that fatty droplets are sometimes arranged like a belt around the lumen of the vessel. In contrast to the findings in similar material from cases of hypertension, they have found that the fatty droplets are in the inner layer of the vessel wall, leaving the adventitia free. In some instances where the fatty infiltration was of notable degree they found the vessel wall ectatic, actually forming an aneurysm.

A lipotropic hormone believed to be secreted by the alpha cells of the pancreatic islands of Langerhans has been reported. A deficiency of this hormone would appear probable in diabetes and may be responsible for some of the fatty droplets in the vascular tree.[11]

Variation in ocular tension that occurs in diabetes has been considered a possible factor in the production of the diabetic retinal aneurysm.[12] It has been pointed out, however, that these aneurysms are not seen in newly formed, widely dilated retinal capillaries in cases of diabetic retinopathy showing localized retinal venous occlusions. This would seem to indicate that increased capillary pressure in itself is not sufficient to cause the aneurysmal dilatations.[1] It appears that diabetes mellitus in time produces a weakening of the capillary wall.

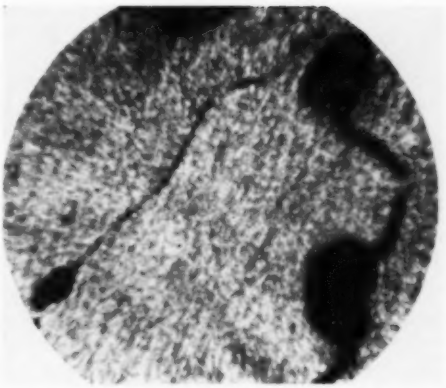

Fig. 9 (Hartford). Lipid material in aneurysmal wall (scarlet red).

Fig. 10 (Hartford). Beading of the venules.

Other vascular changes

In the group of diabetes mellitus cases herein reported, varices of the retinal venules were observed infrequently (fig. 10). It has been found that varices in the retina are seen occasionally in patients with diabetes mellitus and more rarely in those with arteriosclerotic retinal changes.[13]

Phlebosclerosis is not outstanding in this series of retinas. In many areas surrounding the microaneurysms no change in the venules is noted.

Retinal hemorrhages occur irregularly in the three groups of diabetic cases but are fewer in those without arteriosclerosis or hypertension. Ophthalmoscopically observed white plaques have been shown to consist of an albumin-rich extravasation into the internuclear layer of the retina with essentially no fibrin masses, cellular elements, or fat[2] (fig. 11). In many cases patients do not complain about loss of vision, for hemorrhages or exudates frequently do not involve the macular area.[14] Exudates are often lacking until several years after the emergence of varicose buds and punctate hemorrhages.[15]

Of the diabetic cases herein reported, microaneurysms are present in all but two cases. Table 1 summarizes the cases in this series in which the duration of the diabetes was known. Duration for one of the two patients in whom microaneurysms were not found was apparently short but unknown.

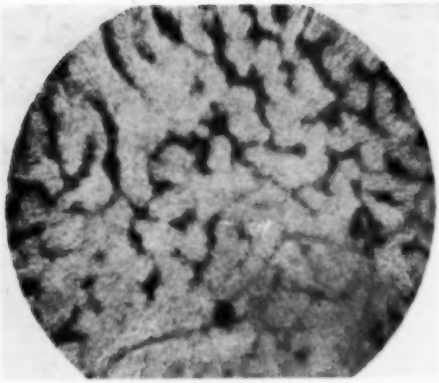

Fig. 11 (Hartford). Deposits of inspissated exudate.

For the other case, the disease had been known to exist for 11 years. With dietary control, which apparently was intermittently satisfactory, there had been no glycosuria during much of the time. In none of these 21 cases was the control of diabetes consistently satisfactory. Patients became careless about diet and frequently neglected insulin. The majority of these patients had long-standing diabetes. In them, retinal lesions were found with greatest frequency. (The renal lesions of Kimmelstiel-Wilson[9, 16] were observed in two thirds of the cases.)

Determent of vascular changes

Perseverance on the part of the patient and his doctor in the management of diabetes mellitus is most important. Assurance of proper calorie intake (20 percent of which is supplied by protein), uniform and adequate insulin supply, and maintenance of freedom from obesity are considered proper management at this time. Control of capillary fragility is an additional important factor.[17] Diabetic camps have proved useful in supporting the morale of diabetic adolescents.

A group of patients with diabetes carefully controlled for 20 to 33 years have been reported to have normal vision. The report stresses that these patients faithfully followed their diabetic regimes, avoiding obesity, glycosuria, hyperglycemia, high-fat or high-calorie diets.[18] Joslin has shown that strict treatment offers the diabetic a good prognosis for general health.[19]

Control group

Retinas from 21 cases in which diabetes was not known were examined in this series. No aneurysms were found in the capillary beds of 15 (fig. 12). In five cases, a scant number of isolated aneurysms were noted. In the sixth case, several groups of aneurysms were present. Some of these were large and had the appearance of those found in diabetes. No glycosuria had been found in this patient. The blood-sugar level had not been determined.

TABLE 1

SUMMARY OF CASES IN WHICH THE DURATION OF THE DIABETES WAS KNOWN

No. of Cases	Known Duration of Diabetes Mellitus (years)	Retinal Microaneurysms	Inter- or Intra-Capillary Glomerulosclerosis
1	20	Present	Present
1	17	Present	Present
1	14	Present	Present
1	12	Present	Present
4	11	Present in 2 Absent in 1 Scant in 1	Present in 1 Absent in 3
2	10	Present	Present
2	9	Present	Present
2	7	Present	Present in 1
1	4	Present	Present
6	Unknown	Absent in 1	Absent in 3

It has been found that, in diabetes, the blood-sugar level can remain above the high limit of normal and yet be sufficiently below the renal threshold so that glycosuria does not occur. Even the constant presence of hyperglycemia appears to be unnecessary in the development of diabetic retinal changes in those occasional cases in which they are present without glycosuria or hyperglycemia but in which definitely positive glucose-tolerance curves are present and in which definite clinical evidence of diabetes may appear at a later date.[3]

In a large series of retinas obtained at autopsy and enucleation from cases in which diabetes mellitus was not known, Ashton[11] found microaneurysms in 33 percent of the former and 33.7 percent of the latter. The greater number of these retinas showed only one or a few aneurysms and no lesions suggestive of microaneurysms clinically.

In his series of nondiabetic cases, Ashton did not ignore the finding of one or two aneurysms. He reported that nondiabetic aneurysms are frequently small and located peripherally, and concluded that diabetic retinopathy remained the condition in which microaneurysms are seen most frequently and in their greatest numbers and most advanced forms.

In hypertensive vascular disease, dilatation and sacculation of capillaries toward the arteriolar side and focal reduplication of basement membranes have been reported to be the essential lesions.[20] In the early phase of diabetic retinopathy, the microaneurysm is considered to be a primary vascular change. When it is found in nondiabetic retinas, it is thought to be a secondary vascular change. Some of the capillary wall thickenings contain lipid material.

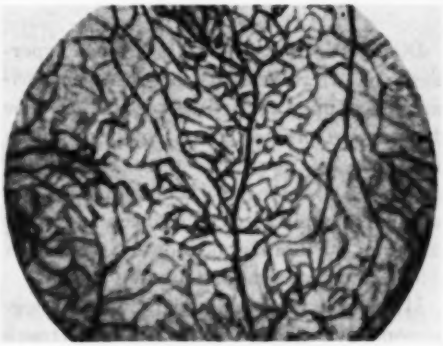

Fig. 12 (Hartford). Capillary bed with no aneurysms.

The age of the diabetic patients examined (table 2) was predominantly between the 45th and the 65th years; in the 21 cases in which no glycosuria was present, there was a wider age distribution.

Table 3 shows the number of cases in both the diabetic and control groups in which microaneurysms were found. One of the 21 control cases is a probable case of diabetes mellitus (mean diastolic pressure over 95 mm. Hg). Glucose tolerance-curve determinations do not positively rule out the disease in the control cases.

TABLE 2

AGE DISTRIBUTION OF PATIENTS

Age Group (years)	Diabetics	No Known Diabetes
26 to 35	0	1
36 to 45	0	3
46 to 55	5	4
56 to 65	11	4
66 to 75	2	3
76 to 85	2	6
86 to 95	1	

TABLE 3

KNOWN DIABETES MELLITUS (21 CASES)

	No arteriosclerosis No hyperpiesia (6)	Arteriosclerosis (No hyperpiesia) (8)	Mean Diastolic Pressure over 95 mm. Hg (7)
Microaneurysms	6/6	6/8	7/7

NO KNOWN DIABETES MELLITUS (21 CASES)

	No arteriosclerosis No hyperpiesia (7)	Arteriosclerosis (No hyperpiesia) (6)	Mean Diastolic Pressure over 95 mm. Hg (8)
Microaneurysms	1/7	1/6	4/8

Summary

Diabetes mellitus of itself, without hyperpiesia or arteriosclerosis, produces deleterious effects upon the retinal vasculature. The changes are predominantly in the capillary tree and are more numerous toward the venous side. Venous dilatation and tortuosity is found in a minority of instances. Increased caliber of the capillary bed toward the venous side is found only in local areas.

Microaneurysms, although not pathognomonic of diabetes mellitus, are found much more frequently and in greater numbers in the retinas of patients with long-standing cases of this disease. They are found in the presence of patent connecting capillary and venular channels, and occur in any quadrant of the fundus. Some appear to be the sites of hemorrhages.

Weakening of the basement membrane of the capillaries from a biochemic change in its carbohydrate-protein components is postulated as preceding aneurysmal formation. Thickening of the wall of an aneurysm, on one side most remote from the main flow through the capillary, is noted. Staining indicates that the chemical composition varies in these thickenings—carbohydrate and lipid materials are demonstrated.

Perseverance in maintaining day-to-day optimum conditions for the patient with diabetes mellitus is noted by some investigators to minimize the deleterious effects that the long duration of this disease has upon vision.

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Acknowledgment is made to Dr. Donald J. Lyle for his advice and encouragement, to Miss Eileen McGreevy for her assistance, and to the Department of Pathology for making this study possible.

References

1. Friedenwald, J. S.: Diabetic retinopathy. Am. J. Ophth., **33**:1187 (Aug.) 1950.
2. Wagener, H. P.: Retinopathy in diabetes mellitus. Proc. Am. Diabetes A., **5**:203, 1945.
3. Elwyn, H.: Problem of diabetes retinitis. Arch. Ophth., **25**:139 (Jan.) 1941.
4. Ballantyne, A. J.: Retinal changes associated with diabetes and with hypertension: A Comparison and contrast. Arch. Ophth., **33**:97, 1945.
5. Gifford, S.: Symposium on diabetic retinitis. Am. J. Digest. Dis., **10**:329 (Sept.) 1943.
6. Friedenwald, J. S.: A new approach to some problems of retinal vascular disease. Am. J. Ophth., **32**:487 (Apr.) 1949.
7. Agatston, S. A.: Clinicopathologic study of diabetic retinae. Arch. Ophth., **24**:252 (Aug.) 1940.
8. Day, R.: Polysaccharides in ocular tissue. Am. J. Ophth., **33**:224, 1950.
9. Schneider, R. W., Lewis, L., Moses, J., and McCullagh, E. P.: Retinal hemorrhage and lens changes in alloxan diabetes in rabbits. Proc. Cent. Soc. Clin. Res., **17**:17, 1944.
10. Ballantyne, A. J., and Lowenstein, A.: The pathology of diabetic retinopathy. Tr. Ophth. Soc. U. Kingdom, **63**:95, 1943.
11. Ashton, N.: Retinal microaneurysms in the nondiabetic subject. Brit. J. Ophth., **35**:189 (Apr.) 1951.
12. ———: Vascular changes in diabetes with particular reference to the retinal vessels. Brit. J. Ophth., **33**:407, 1949.
13. O'Brien, C. S., and Allen, J. H.: Unusual changes in retinal veins in diabetes. Arch. Ophth., **24**: 742 (Oct.) 1940.
14. Croom, J. H., and Scott, G. I.: Retinal and vascular damage in long-standing diabetes. Lancet, **1**:555, 1949.
15. Doggart, J. H.: Retinopathy in the diabetic. Tr. Ophth. Soc. of Australia, **9**:139, 1949.
16. Waite, J. H., and Beetham, W. P.: The visual mechanism in diabetes mellitus (a comparative study of 2,002 diabetics and 457 nondiabetics for control), New England J. Med., **212**:367, 429, 1935.
17. White, P.: Free vs. controlled regime in juvenile diabetes. J. Am. Dietetic A., **25**:757, 1949.
18. Sherrill, J. W.: Diabetic retinitis: The relationship between retinal degenerative changes and the degree of diabetic control. Bull. Scripps Metabolic Clinic, **2**: (Oct.) 1951.
19. Joslin, E. P.: Status of living diabetics with onset under forty years of age. J.A.M.A., **147**:209 (Sept.) 1951.
20. Wexler, D., and Branower, G.: Retinal capillary lesions in malignant hypertension. Arch. Ophth., **44**:539, 1950.

CENTRAL RETINAL VEIN THROMBOSIS*

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During the past few years several papers on central retinal vein thrombosis have appeared in the ophthalmic literature. Some of these have favored anticoagulant therapy,[1-5] while others[6-8] have not believed anticoagulants effective in relieving local thrombosis of retinal veins.

To study this subject further, I am reviewing 54 cases of central retinal vein thrombosis that I have seen during the past 10 years. In addition to anticoagulant therapy, the onset of thrombosis, the relationship to the general physical condition, the visual acuity after the thrombosis, and the final vision have been reviewed. The appearance of the retina, the method of clearing, the life expectancy after this episode, and the secondary hemorrhagic glaucoma which so frequently follows central retinal vein thrombosis have been considered together with the prognosis for the affected eye.

Symptoms

Central retinal vein thrombosis is a clinical entity in which a thrombus forms in the central retinal vein, the lumen of which has been partially obstructed by obliterative endophlebitis. The close association of the retinal artery and vein in a common sheath results in a partial obstruction of the vein from a sclerosis of the adjacent artery. Stasis of the blood flow is followed by partial obstruction and then by complete venous obstruction.

The symptoms of the onset of retinal vein thrombosis would indicate that the occlusion could develop gradually in some cases and suddenly in the others. Photopsia and dimmed vision preceded the completely blurred vision that occurred with the thrombosis in nine of the 54 cases. Some inciting factors other than arteriosclerosis and hypertension seemed to cause the thrombosis in several instances.

Inciting factors

Two cases occurred in diabetics, one in a patient with polycythemia vera, and one case followed a severe influenza. Two cases occurred after trauma, one from the explosion of a light bulb close to the face, and the other from a blow in the face with a timber. One case followed a severe vomiting attack and one the injection of varicose veins.

All of the other cases occurred in patients with marked arteriosclerosis or hypertension or both. Fright, severe exposure, and emotional upsets were cited by several patients as being the inciting factor but this was rather difficult to prove. The general condition of the patient, the heart, the kidneys, the arteriosclerosis or hypertension were the important factors.

Inciting factors

Patient	Incident
W. F.	Bulb exploded in face
L. A.	Struck in face with timber
L. M.	Diabetic retinitis
M. K.	Severe vomiting
C. F.	Influenza
R. E.	Diabetic retinitis
A. I.	Polycythemia vera
G. M.	Varicose vein injection

Early visual acuity

Theoretically, the primary visual acuity would depend upon the site and extent of the thrombosis but it doesn't seem to follow such a clear-cut pattern. In some instances, a branch thrombosis caused as much or more visual disturbance than a complete thrombosis.

The macular involvement, together with the amount of blood that perfuses the retina, and the amount of arteriosclerosis present in

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the retinal arteries are determining factors. The visual acuity depends upon the site and extent of the disturbance of the retinal metabolism.

EARLY VISUAL ACUITY

NUMBER OF PATIENTS	EARLY VISUAL ACUITY
5	20/20 to 20/40
6	20/40 to 20/70
6	20/70 to 20/200
20	20/200 to H.M.
11	Hand movements
5	Light perception

In 11 patients (20 percent) the visual acuity was serviceable, but in 43 patients (80 percent) visual acuity varied from industrial to complete blindness.

HYPERTENSION AND ARTERIOSCLEROSIS

As has been previously stated, thrombosis of the central retinal vein ordinarily results from hypertension or from arteriosclerosis. Hypertensive and arteriosclerotic changes in the vessels of the opposite eye were found in 17 of the 54 patients. Two of these had had previous attacks of retinal thrombosis of the other eye.

The blood pressure in most instances was a good criterion of the prognosis, the higher the blood pressure the worse the ultimate result. The blood pressure in this group was elevated in 37 cases, the diastolic in most instances being much higher than normal.

Arteriosclerotic changes in the opposite eye were present in some with and in others without hypertension. Diabetes as previously mentioned apparently predisposes in some instances to retinal vein thrombosis. Both of the diabetic patients, however, also had hypertension.

EYE FINDINGS

The involved eye showed papilledema and engorged tortuous veins while retinal hemorrhages and extravasations of exudate were interspersed over the retina, hiding the vessels and much of the retina. The extravasated blood was preretinal in three instances and in two was in the vitreous. Branch thrombosis was seen in 12 cases but in all of these there was involvement of the macula and the visual impairment was just as severe as if the thrombosis had been complete.

CLINICAL COURSE

The clinical course was introduced by the sudden appearance of hemorrhages and papilledema. Tortuous veins and exudate were present. In some cases the eyes were seen within a few hours after the thrombosis started. In no instance did the process become more extensive. It remained stationary for a long time and did not seem to improve.

Eventually, in four to eight months, the papilledema diminished, the hemorrhages became lighter in color, while the white exudates seemed to be more in evidence. The tortuosity of the veins remained as did the attenuation of the arteries and the sheathing or obliteration of the vessels. The discs acquired many new vessels and capillaries. In some eyes, new, small capillaries appeared above or below the macula.

Reëstablishment of circulation with these new capillaries or peripapillary vessels did not appear until after the papilledema had disappeared. The new vessels were usually small and closely packed and nearly covered the surface of the disc. In two or more instances, similar vessels were found in both eyes, although the thrombosis had only been in one eye.

Foster Moore believed these loops of vascular anastomosis which can be seen on the papilla were congenital malformations. In two of my cases, however, I am sure that they did not develop until after the thrombosis had occurred and had begun to clear.

RETINAL CHANGES

Ultimate retinal changes consisted, in most instances, of perimacular degeneration with depigmentation around the macular region. In six (13 percent) of the 46 cases which did not go on to hemorrhagic glau-

coma, a marked whiteness of the optic disc or optic atrophy followed the central retinal vein thrombosis. Perimacular depigmentation was seen in 11 (25 percent) of the 46 cases.

Final visual acuity

In spite of a lack of visual evidence of marked changes in the retina, functional recovery of vision is extremely unlikely. In these 54 cases of central retinal vein thrombosis, five had a final visual acuity of 20/20 to 20/30; five had 20/50 to 20/70; eight had vision of 20/200; while the remainder had from 20/300 to total blindness.

Final visual acuity

Number of Patients	Percent	Visual Acuity
5	10	20/20 to 20/30
5	10	20/50 to 20/70
8	16	20/200
12	25	20/300 to 20/400
15	30	C.F. to H.M.
8	16	Blind or enucleate

In other words 20 percent of these patients had useful vision and 80 percent were blind in the affected eye. The final visual acuity was not dependent upon the completeness of the central retinal vein thrombosis nor upon anticoagulant therapy but more upon the damage to the macula or optic nerve and upon the interference with retinal metabolism.

Life expectancy

Jansen reported that the average life expectancy after central retinal vein thrombosis was five to six years. When this group of 54 patients seen during the past 10 years was sent follow-up letters requesting reëxamination, I was surprised to find that 18 (33⅓ percent) of the 54 were dead.

Treatment

The treatment used was general hygienic rules to improve general health such as rest, attention to hypertension or diabetes or other systemic disease, and the use of such vasodilators as nicotinic acid, priscol, rutin, and vitamin P, as well as other indicated therapy.

Anticoagulant therapy was used in 10 of these 54 cases.

Anticoagulant treatment
10 Patients

Duration of Treatment (weeks)	Number of Patients	Final Visual Acuity
2	3	H.M., 20/100, 3/200
3	1	6/200
4-5	2	3/200, C.F.
12-15	2	20/200, 20/60
52 or more	2	1/200, 3/200

The shortest period anticoagulant therapy was used was two weeks, the longest 16 months. The internists were loathe to keep these 10 patients using anticoagulants under treatment for any length of time. I appreciated their alarm after one patient from the group had intestinal hemorrhage, hematuria, and hematemesis. In all of this group of 10, anticoagulant therapy was started relatively early and only two patients retained or recovered better than 20/200 vision; one retained 20/60, which she had when first seen with the retinal vein thrombosis; the other recovered final acuity of 20/100. The other eight patients were given heparin or dicumarol or both and either did not improve or became worse.

Prognosis

The prognosis of the central retinal vein thrombosis in this series was not influenced by any treatment. The only patients who improved were the ones who were young and had no appreciable hypertension. Two who were in the thirties and one without hypertension, in his fiftieth year, had fair functional recovery. If they were older or if they had hypertension, the retinal function was destroyed by the thrombosis.

Hemorrhagic glaucoma

Hemorrhagic glaucoma occurs so frequently after central retinal vein thrombosis it seems to be a further development of the pathologic changes initiated by the thrombosis. In these 54 cases, hemorrhagic glaucoma developed in nine (16 percent). The

latent period between the thrombosis and the inception of a hemorrhagic glaucoma varied from six weeks to three years, the average time about three or four months.

These nine patients with hemorrhagic glaucoma suffered pain, unilateral headache, nausea, and vomiting. In the cases followed, the intraocular pressure was low immediately after the thrombosis, then it seemed gradually to rise to readings of 30 to 40 mm. Hg and even higher. Miotics did not appreciably influence the tension even when miotics were started as soon as it began to rise.

The gradual development of circumpupillary vascularization was associated with a steamy cornea. A brownish precipitate, suggesting an advanced depigmentation, almost a uveitis type of exudate, appeared on the posterior corneal surface.

The vascularization of the iris progressed. A mild ciliary injection appeared. The pupil was enlarged and immobile. Hyphema occurred in four of the nine cases. The ocular tension was very high, the cornea edematous, and light perception was absent.

In two of these nine cases enucleation was avoided but the eyes developed bullous keratitis, followed later by hypotension and phthisis bulbi with cloudy vascularized corneas. Six of the nine patients required enucleations. Preliminary filtering operations or cyclodiathermy was tried but in only one patient was the cyclodiathermy operation effective in reducing the tension enough to save the globe.

The prognosis in hemorrhagic glaucoma is always so grave it is practically hopeless to anticipate saving the eyeball. If the eye with hemorrhagic glaucoma is seen without the preceding thrombosis having been observed, the danger of an intraocular tumor should influence the decision to enucleate rather than to resort to any temporizing measure to reduce tension. Blindness and a painful eye are certain and enucleation is the treatment of choice.

One theory of the pathogenesis of hemorrhagic glaucoma is that it results from a toxin which causes inflammation of the chamber angle producing a vascular membrane of the angle and iris. It has been postulated that, if glaucoma precedes the thrombosis, hemorrhagic glaucoma will be more likely to occur. In two of my 54 cases of thrombosis of the central retinal vessels, glaucoma was present but hemorrhagic glaucoma did not occur.

Sclerosis of the uveal vessels and recurrence of the thrombosis have been suggested as hypotheses to account for the pathogenesis of hemorrhagic glaucoma. No satisfactory explanation has been advanced.

In hemorrhagic glaucoma, there is gradual elevation of the intraocular pressure, irreversible dilatation of the pupil, pain, and blindness; then the symptoms subside and there is a drop in the tension and degeneration of a soft eye. Subsidence of symptoms is seldom seen because their severity usually necessitates enucleation. The tension often remains high with a subsidence of the pain and acute symptoms. I have seen two such eyes in which tension was high yet there was no pain or other symptoms except blindness.

Conclusion

In this series of cases, central retinal vein thrombosis usually occurred in older persons with hypertension. The onset was sudden but often there was some premonitory visual disturbance.

Emotional upsets, trauma, systemic diseases, or vasomotor disturbances may have been contributory factors in some cases.

Papilledema, retinal hemorrhages, exudates, and tortuous veins were found in the affected eyes. Reëstablishment of retinal circulation did not affect the profound loss of vision.

Anticoagulant therapy, when used, did not appreciably influence either the retinal appearance or the recovery of function. There was no appreciable improvement in vision with or without anticoagulant therapy. Eighty percent of the patients remained blind.

Hemorrhagic glaucoma occurred in nine

of the 54 cases or in 16 percent. In all of these, vision was lost. Six of the nine required enucleation to alleviate the stormy symptoms.

The prognosis of central retinal vein thrombosis is extremely grave and shows a direct relationship to the age and hypertension of the patient.

There is no effective treatment.

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References

1. McLean, J. M., and Puig Solanes, M.: Anticoagulant therapy in retinal vascular occlusion. Tr. Am. Acad. Ophth. July-Aug., 1949, pp. 644-657
2. McLean, A., and Brambel, C. E.: Dicumarol and rutin in retinal vascular disease. Tr. Am. Ophth. Soc., **44**:194-213, 1946.
3. Klien, B. A.: Anticoagulant therapy of occlusion of central vein of retina: In relation to pathogenesis and differential diagnosis. Arch. Ophth., **29**:699-710 (May) 1943.
4. Pagliarani, N.: The treatment of thrombosis of the retinal veins with anticoagulants. Giornale Italiano di Oftalmologia, **2**:335-349 (Sept.-Oct.) 1949. (Abstract: Am. J. Ophth., **33**:1479, 1950.)
5. Ploman, K. G.: Treatment of thrombosis of the retinal veins with heparin. Acta Ophth., **21**:190-203, 1943.
6. Braendstrup, P.: Central retinal vein thrombosis and hemorrhagic glaucoma. Acta Ophth., Supp. 35, 1950, pp. 1-162.
7. Larsson, S., and Nord, B.: Some remarks on retinal vein thrombosis and its treatment with anticoagulants. Acta Ophth., **28**:187-201, 1950.
8. Huggett, A. St.G., and Juler, F. A.: Heparin in thrombosis of central vein of retina. Tr. Ophth. Soc. U. Kingdom, **62**:123-134, 1943.

DIAGNOSTIC SIGNIFICANCE OF RETINAL ARTERY PRESSURE IN INTERNAL CAROTID INVOLVEMENT*

Findings in eight cases with carotid-artery lesions compared to 50 controls and 210 cases from the literature

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Comparison of retinal artery pressure in the two eyes affords a readily available diagnostic tool that is helpful in neurologic diagnosis when cerebral symptoms are suspected of being secondary to lesions of the internal carotid artery.

In four of our five cases of proven carotid-artery thrombosis and three of carotid ligation, all with cerebral symptoms (Thomas,[1] 1949, 1950), the retinal artery pressure was found to be significantly lower on the side of the obstruction. Observations on 50 normal subjects and patients with other illness have been made (100 eyes examined to evaluate the significance of this finding).

In the literature, the findings in all cases in which bilateral retinal-artery pressures have been recorded (199) have been reviewed and included as controls.

Discussions of the significance of retinal-artery pressures in hypertension, increased intracranial pressure and other conditions, have in general neglected consideration

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of the diagnostic implications of differences between the two eyes (Duke-Elder,[2] 1926; Koch,[3] 1945; Bailliart,[4] 1928, 1938, 1947; Espildora-Luque,[5] 1947; Luo,[6] 1939; Bardram,[7] 1944; Berens,[8] 1928; Uyemura,[9a] 1941; Constantine,[10] 1940; de Sanctis,[11] 1938; Franceschetti,[12] 1949; Gutmann,[13] 1943; Kapuscinski,[14] 1948; Lebensohn,[15] 1925; Linksz,[16a] 1939; Magitot,[17] 1919, 1922; de Morsier,[18] 1939; Streiff,[19a-c] 1937, 1941, 1942). In recent years a few reports have stressed this, particularly in carotid lesions (Krayenbuhl,[20] 1935; Schiff,[21] 1944; Miletti,[22a-b] 1946, 1947). Observations in 11 such cases are reviewed.

Method of study

Subjects

The subjects examined were: (1) Healthy adults and hospital patients without evidence of neurologic disease; (2) patients with evidence of neurologic disease; and (3) patients with carotid-artery thrombosis or ligation.

Technique

The following routine was employed in the study of these subjects: (a) Diastolic and systolic retinal-artery pressure was measured by Bailliart's method (Bailliart,[4a] 1928; Lebensohn,[15] 1925; Friedenwald,[23] 1934); (b) in part of the cases the right eye was examined first, and vice versa; (c) examinations were done under mydriasis with 0.5-percent homatropine solution and local anesthesia with 0.5-percent pontocaine solution, with pontocaine alone, or with no preparation of the patient; (d) blood pressures in each brachial artery were taken for comparison at the same time, just before the retinal-artery pressure was checked or immediately after. The position of the patient at the time of the examination was recorded.

The central retinal artery arises from the ophthalmic artery, which branches from the internal carotid. Hence determination of the retinal artery pressure gives an indication of blood flow near the terminal end of the internal carotid artery, thus offering a simple nontraumatic means of investigation of these vessels.

A measure of the retinal-artery pressure (R.A.P.) is obtained by observing with direct ophthalmoscopy the appearance and disappearance of pulsations of the central retinal artery when external pressure is applied to the globe with the blunt end of the plunger of Bailliart's[4] ophthalmodynamometer. The latter is a simple cylindrical apparatus which has a spring resistance and is calibrated to measure pressures ranging from 10 to 150 grams of water.

Besides Bailliart's instrument there have been other similar apparatuses such as those of Sokanski,[24] Uyemura and Suganuma,[9b] Kukan,[25] Keil,[26] Linksz,[16b] and Gutmann.[13] Most of these are modifications of Bailliart's type of apparatus which was used in the present study.

The examination can be done with the patient in either the upright or recumbent position. A slight degree of mydriasis is advisable, which may be produced by using a two-percent solution of ephedrine or 0.5-percent solution of homatropine. Local anesthesia with pontocaine (0.5-percent) is necessary only with nervous and uncoöperative patients.

To examine the right eye the ophthalmoscope is held as usual in the right hand. The ophthalmodynamometer is held horizontally between the thumb and forefinger of the left hand. Positions and hands are reversed to examine the left eye.

The rounded end of the pressure gauge is introduced between the lids near the external canthus and applied to the sclera near the insertion of the lateral rectus muscle. The figures marked on the rod of the apparatus should be turned toward the examiner.

When the observer obtains a perfect picture of the fundus with direct ophthalmoscopy, his attention is turned to the disc where the pulsation of the vessels is clearly

visible. Then he begins to press the eyeball with the blunt end of the rod, watching for the first pulsation of the central retinal artery (or its papillary branches), which appears when the diastolic arterial pressure is reached.

As soon as he sees the first pulsation he must hold his hand perfectly still while either he or his assistant makes the readings on the scale of the instrument. The examiner then increases the external pressure until the visible arterial pulsation has ceased. The systolic pressure corresponds to the last arterial beat. He then notes the grams of pressure indicated on the scale.

A few points of ophthalmodynamometry should be emyhasized: (1) The apparatus should be held perfectly horizontal during the examination so that its own weight does not affect the results; (2) it is important to work rapidly and to observe exactly the first pulsation of the central retinal artery, which indicates the diastolic level, and then to continue quickly to reach the systolic pressure.

This is the crucial reading, because the pulsation tends to reappear if the instrument is retained on the sclera for a few seconds without reducing the compression. The reappearance of the arterial pulsation is due to the reduction of the intraocular pressure from the scleral compression.

It is known that the more one presses on the eyeball the more the tension falls. The ocular hypotony disappears after 20 to 30 minutes, therefore, if the crucial reading is missed, a second observation must be delayed for at least that long to avoid false readings (Bárány,[27] 1946; Magitot,[17b,c] 1919, 1922; Lebensohn,[15] 1925).

Ophthalmodynamometry is a simple technique. In most of our cases, it took only about as long as checking the blood pressure and was no more disturbing to the patient. One observer (M. P.) usually made the readings without knowing the nature of the neurologic disease. The pertinent difference between the grams of pressure on the two eyes was selected as the most convenient manner of comparison for each case.

$$\frac{\text{Higher R.A.P.} - \text{Lower R.A.P.}}{\text{Higher R.A.P.}} = \% \text{ difference}$$

Conversion scales are available for changing the recorded pressures from grams of water to millimeters of mercury (Magitot,[17b] 1919), but for our purpose of simply comparing the two eyes these were not used.

Objections have been made to the use of Bailliart's[4] method of ophthalmodynamometry as a quantitative measure of actual lateral pressure in the retinal arteries (Duke-Elder,[2] 1926; Koch,[3] 1945). However, it is obvious that most of these objections do not apply if (1) the same observer examines both eyes in rapid succession, (2) brachial blood pressures are checked at the time of examination, (3) the same instrument is used for successive readings. The mean error in a large series of double determinations has been found to be only two to four grams between readings (Strieff,[19d] 1946). Differences in intraocular pressure may be detected by palpation or by using the Schiøtz tonometer.

Results in controls

Retinal artery pressures in healthy young adults and in patients without evidence of neurologic disease (16 individuals) are shown in Table 1. Findings in 34 patients with neurologic disease not resulting from carotid-artery lesions are shown in Table 2.

Review of our observations in these 50 cases indicates that the following factors do not appear to affect the readings as regards the equality of pressures: (1) Use of homatropine, pontocaine, or no preparation at all; (2) which eye was examined first; or (3) the position of the patients.

In uncoöperative or excitable patients, results were not considered reliable when delay in determining the end-point introduced an error as a result of sustained compression of the globe (Espildora-Luque,[5] 1947; Bailliart,[4a] 1928; Lebensohn,[15] 1925; Luo,[6] 1939; Magitot,[17c] 1922).

TABLE 1

RETINAL ARTERY PRESSURES IN HEALTHY ADULTS AND PATIENTS WITHOUT NEUROLOGIC DISEASE

Case No.	Diagnosis	Brachial Blood Pressure		Retinal Artery Pressure					
				Diastolic			Systolic		
		Right (mm. Hg)	Left	O.D.	O.S.	Diff. (percent)	O.D.	O.S.	Diff. (percent)
1	Normal	126/86	124/90	18	18	0.0	90	100	10.0
2	Normal	110/70	110/74	25	19	16.0	100	100	0.0
3	Normal	108/66	110/68	10	10	0.0	68	64	5.9
4	Normal	114/70	98/68	5	5	0.0	49	37	24.5
5	Normal	124/76	126/78	22	22	0.0	76	78	2.5
6	Normal	118/78	110/75	5	20	75.0	90	82	8.8
7	Fracture of femur, bilateral	120/75	120/80	17	22	22.8	72	76	5.7
8	Hypertension	176/125	178/128	94	93	1.1			
9	Disseminated vascular disease	118/80	100/70	28	23	17.9	78	69	11.5
10	Hyperthyroidism	114/68	112/64	20	28	28.6	128	122	4.7
11	Chronic cholecystitis	154/82	150/84	20	28	28.6	130	138	5.8
12	Rheumatic heart disease	100/80	98/75	16	12	25.0	49	43	12.2
13	Hypertension	165/108	165/110	93	79	15.0			
14	Postthoracoplasty for pulmonary tuberculosis	186/100	130/70	120	40	66.6			
15	Psychoneurosis	150/75	150/74	15	18	16.6	98	89	9.2
16	Psychoneurosis	126/86	126/85	18	27	33.0	101	107	5.6
Excluding cases with diastolic pressure of 20 gm. or less or brachial pressures differing by 15 mm. Hg or more:									
Average percent difference						2.7%			6.4%
Range of percent differences				0–15%			0–12.2%		

Asymmetry of systemic blood pressure as commonly measured in the brachial vessels (Kerr,[28] 1936) would naturally be shown in inequality of the retinal artery pressures. Our material demonstrated this inequality to a marked degree in one case following thoracoplasty and to a lesser extent in three other controls. To eliminate this source of inequality, cases with 15 mm. mercury or more difference in brachial blood pressures have been omitted in computing the range of variation and average percent difference between the two eyes shown in Table 3.

Likewise individuals with low diastolic pressures were excluded because, with the equipment used, values of 20 gm. diastolic pressure and below were found to be quite variable on repeated examinations. Also, relatively minor numerical differences in this range are given disproportionate emphasis by the percent difference comparison. For example, a five-gm. difference constitutes 25-percent difference between 15 gm. and 20 gm. but only five-percent difference in the 95- to 100-gm. range.

Findings in patients with cerebral and noncerebral lesions were essentially the same so they have been grouped together in Table 3.

Comparison of our observations with those gleaned from the literature is made in the next two tables. Table 4 shows the findings in 106 normals reported and Table 5 gives the data on 93 patients found in the literature in whom bilateral determination of the retinal artery pressure was made. These include a wide range of neurologic and nonneurologic pathologic conditions, such as hypertension, central nervous-system lues, and brain tumors. A single case (de Morsier,[18] 1939) was excluded, since the history and findings were strongly sug-

TABLE 2

RETINAL-ARTERY PRESSURES IN PATIENTS WITH NEUROLOGIC DISEASE NOT RESULTING FROM CAROTID LESIONS

Case No.	Diagnosis	Brachial Blood Pressure (mm. Hg)		Retinal Artery Pressure					
				Diastolic			Systolic		
		Right	Left	O.D.	O.S.	Diff. (percent)	O.D.	O.S.	Diff. (percent)
1	Convulsive disorder	118/84	114/80	64	70	8.2	122	128	4.7
2	R. hemiparesis, aphasia			60	62	3.3	135	132	3.2
3	L. parietotemporal neuroblastoma			41	42	2.4	92	105	12.4
4	Headache, functional	100/75	106/75	15	12	20.0	55	50	9.1
5	Post. inf. cerebellar artery thrombosis	142/110		71	72	1.4	123	135	5.9
6	Cerebral arteriosclerosis and hypertension	143/105	158/110	39	46	15.2	121	140	13.6
7	Multiple sclerosis	105/70	104/75	28	24	14.3	43	43	0.0
8	Cerebral thrombosis with R. hemiplegia	150/90	150/90	58	52	10.0	128	148	13.5
9	Convulsive disorder and psychosis	88/50	100/50	17	19	10.1	59	56	5.1
10	Post. fossa meningioma	138/94	142/98	59	57	3.4	140	145	3.8
11	Spastic tetraparesis			18	15	16.7	82	90	8.9
12	Migraine	100/68	98/68	12	10	16.7	54	62	12.9
13	Congenital diplegia with convulsive disorder	124/82	130/88	38	37	2.6	118	128	7.8
14	Cervical myelitis	138/80	128/95	10	40	75.0	105	102	2.6
15	Bulbospinal degeneration	130/90	126/86	22	23	4.3	80	86	7.5
16	Paraplegia	154/92	148/90	35	38	7.9	131	128	2.3
17	Conversion hysteria	110/68	110/68	5	5	0.0	43	46	6.5
18	Multiple sclerosis and hypertension	205/140	205/140	81	83	2.4			
19	Paraplegia	110/78	108/80	16	18	11.1	61	58	4.9
20	Spinal cord tumor	115/80	112/78	38	32	15.7	95	96	1.0
21	Cerebral thrombosis with L. hemiplegia, aortic insufficiency	165/80 30–60	165/80 30–60	0	0	0.0	133	132	0.7
22	Headaches, cause undet.	128/78	128/80	28	33	15.1	105	109	3.7
23	Meniere's syndrome	128/80	134/82	35	40	12.5	140	138	1.4
24	Convulsive disorder	95/70	95/65	18	20	10.0	54	60	10.0
25	Aneurysm L. post. commun. artery; 3rd N. palsy	100/70	110/78	10	10	0.0	62	61	1.6
26	Multiple sclerosis	110/75	110/70	18	20	10.0	71	69	4.2
27	Suprasellar dermoid cyst	142/90	155/100	83	85	2.4	141	130	7.1
28	Aphasia; probable embolism	136/94	136/94	34	38	10.5	116	128	9.4
29	Headache, cause undet.	144/100	140/98	15	15	0.0	108	116	6.9
30	Amyetrophy lateral sclerosis	122/85	120/85	18	22	18.2	83	93	10.7
31	L. frontal meningioma	130/85	128/80	71	70	1.4	115	118	2.6
32	Prog. muscular dystrophy	110/80	100/70	7	23	70.0	68	71	4.2
33	Cerebral atrophy, generalized	140/92	140/95	29	34	14.6	122	112	8.2
34	Cerebral thrombosis, R.	160/110	150/100	45	40	11.1	72	65	10.8
Excluding cases with diastolic pressure of 20 gm. or less or brachial pressures differing by 15 mm. Hg or more:									
Average percent difference						6.5%			6.1%
Range of percent differences				0–15.7%			0–12.9%		

gestive of carotid thrombosis, although this was not definitely proved.

A small number of cases with small absolute differences in low diastolic pressures (5.0 to 10 gm.) were excluded because of our observation of poor reliability in this range (see footnotes). In three cases of marked papilledema, inequalities were reported, though most of such cases fell within the usual range.

Two cases with high or unequal tonometer readings were likewise excluded in computing averages, since the differences might be attributed to ocular pathology rather than to vascular system changes primarily. If bilateral brachial blood pressures had been recorded, as in our series, another source of inequality might have been screened.

A summary of findings in the 249 controls is shown in Table 6.

It will be noted that, in the 199 reported cases and our 50 cases, there is good agreement as to the range and average percent of difference on the two eyes in the absence of carotid artery involvement. Individual cases show differences up to approximately 15 percent, but for the 249 control cases the average percent difference in retinal artery pressures is 5.2 percent for diastolic and 3.3 percent for systolic. This difference between eyes is about the same as that found between successive readings in the same eye (Streiff,[19d] 1946).

RESULTS IN CAROTID-ARTERY INVOLVEMENT

Eight cases of carotid artery involvement

accompanied by secondary signs and symptoms of central nervous system impairment have recently been observed by us (Thomas,[1] 1949, 1950). Four of these were cases of spontaneous thrombosis of the internal carotid, showing hemiparesis as well as severe aphasia in the three with left-sided involvement. One had internal-carotid thrombosis secondary to a carcinoma invading the neck. All were confirmed by exploration or arteriography. In the other three cases common or internal carotid ligation was carried out for control of hemorrhage or arteriovenous fistula. Observations of retinal artery pressure in these eight cases are shown in Table 7.

In seven of the eight cases, either systolic or diastolic retinal artery pressures showed an inequality beyond the degree of variation observed in the control series. These were of the magnitude of 51.9, 21.4, 33.6, 77.5, and 33.3 percent for diastolic, and 32.5, 36.8, 33.9, and 56.9 percent for systolic. In these seven cases, the lower retinal-artery pressure was found on the side of the carotid involvement. The diastolic pressure difference of 21.4 percent represents a difference in relatively high diastolic pressures (70 and 55 gm.) and hence is of more significance than a similar percent difference occurring in the lower diastolic range.

The other figures speak for themselves, as do the average percent differences in these seven cases of 31.7 percent for diastolic and

TABLE 3

RETINAL-ARTERY PRESSURES—RANGE OF DIFFERENCES AND AVERAGE PERCENT DIFFERENCE BETWEEN THE TWO EYES IN 50 CONTROLS

	No. Cases	Range of Difference		Average Percent Difference	
		Diastolic (percent)	Systolic (percent)	Diastolic (percent)	Systolic (percent)
Normal and without neurologic disease	16	0–15.0	0–12.2	2.7	6.4
Neurologic disease not resulting from carotid lesions	34	0–15.7	0–12.9	6.5	6.1
Average range and % difference	50	0–15.4	0–12.6	5.8	6.1

TABLE 4

RANGE AND AVERAGE PERCENT DIFFERENCE IN RETINAL-ARTERY PRESSURE BETWEEN THE TWO EYES IN NORMALS

Author	Year	No. Cases	Range of Difference		Average Percent Difference	
			Diastolic (percent)	Systolic (percent)	Diastolic (percent)	Systolic (percent)
Salvati[39]	1922	9	Pressures equal bilaterally			
Berens[8]	1928	12	0–17.0	0–8.0	5.2	2.7
de Sanctis[11]	1938	14[a]	0–9.3	0–7.0	1.5	0.7
Constantine[10]	1940	38[b]	0–17.2[c]	0–9.4	5.8	4.1
Miletti[22a]	1946	30	Pressures equal bilaterally			
Miletti[22b]	1947	42	0–20.0[d]	0–11.1	6.0	3.4
Average range and % difference		106	0–15.9	0–8.9	5.2	3.2

[a] Three position studies, totaling 42 comparisons.
[b] Excluding one case in low diastolic range.
[c] Case with 17.2 percent difference had unequal tonometer readings (9 and 10).
[d] Including three cases with over 15-percent difference, but with numerical differences of 5.0 to 10 mm. Hg in relatively low diastolic range.

24.1 percent for systolic as compared to 5.2 percent and 3.3 percent in the controls.

In two cases in which the contralateral carotid was compressed, even greater differences in retinal artery pressures were induced (Miletti,[22a-b] 1946, 1947).

In the last case (Case 8), the contralateral retinal artery pressure was lower. However, the brachial pressures were also unequal, with lowering on the side of the hemiplegia, contralateral to the carotid artery thrombosis caused by the carcinoma or radiation therapy. This phenomenon of lowered brachial pressure occurs apparently as a result of the central nervous-system changes in the opposite hemisphere (Kerr,[28] 1936).

It seems logical that the carotid arteries should also be affected and, in our controls, inequality of brachial pressure was always accompanied by inequality of retinal-artery pressure of a comparable magnitude.

Comparison of the ratios of the retinal to the brachial pressures in Case 8 (diastolic 0.286 and 0.278, systolic 0.618 and 0.680) indicates that the retinal-artery pressures at least roughly correspond to the brachial pressures. Hence, this patient apparently developed sufficient late collateral flow on the left to maintain the retinal-artery pressure roughly equivalent to the brachial pressure

TABLE 5

SUMMARY OF RANGE AND AVERAGE PERCENT DIFFERENCE IN RETINAL-ARTERY PRESSURE ON THE TWO EYES IN VARIOUS CONDITIONS NOT INVOLVING THE CAROTID ARTERY

Author	Year	No. cases	Principal Diagnosis	Range of Difference		Average Percent Difference	
				Diastolic (percent)	Systolic (percent)	Diastolic (percent)	Systolic (percent)
Magitot[17]	1919	1[a]	Posthemorrhage blindness	0.0	0- 6.7	0.0	1.0
Berens[8]	1928	7[b]	Brain tumors and C.N.S. lues	0-15.4	0- 9.0	11.8	5.0
Baratta[30]	1938	34	Optic atrophy, neuritis, and papilledema	0-10.5	0- 6.7	3.3	0.9
Sabbadini[21]	1938	3[c]	Hypertension	0-14.3	0- 7.9	10.9	5.3
Morsier[18]	1939	20[d]	Brain tumors	0-16.6	0-11.0	1.9	1.8
Constantine[19]	1939	7[e]	Lues without optic atrophy	0-12.0	0- 6.0	7.7	4.5
		16[f]	Lues with optic atrophy	0- 8.0	0- 7.6	6.9	2.9
		5	Lues with tryparsamide reaction	0-13.0	0- 2.0	6.2	0.9
Average range and % difference		93		0-11.2	0- 7.1	4.9	1.9

[a] 7 determinations.
[b] Excluding 2 cases with papilledema.
[c] Excluding 1 case with history and findings consistent with carotid thrombosis.
[d] Omitting 1 case with low diastolic values.
[e] Omitting 1 case with 4.0 D. papilledema—bilateral brachial pressure not reported.
[f] Omitting diastolic values in 1 case with high tonometer reading bilaterally.

TABLE 6

RETINAL-ARTERY PRESSURE—SUMMARY OF CONTROLS (249 CASES)

	No. Cases	Average Range of Difference		Average % Difference	
		Diastolic (percent)	Systolic (percent)	Diastolic (percent)	Systolic (percent)
Normals from literature	106	0-15.9	0- 8.9	5.2	3.2
Patients from literature	93	0-11.2	0- 7.1	4.9	1.9
Present series	50	0-15.4	0-12.6	5.8	6.1
Total Controls	249	0-14.0	0- 9.0	5.2	3.3

which was found on that side.

In the acute phase of the occlusion, however, it is quite likely that the left retinal artery pressure was significantly lower at the time symptoms of cerebral involvement developed. Serial retinal artery pressure observations would be necessary to evaluate this sequence.

Previous observations on retinal artery pressures in the presence of carotid artery lesions are summarized in Table 8. It is noted that in eight of 11 cases reported, or all cases where carotid ligation was not a factor, significant lowering of the ipsilateral diastolic and systolic retinal artery pressure occurred.

TABLE 7

RETINAL-ARTERY PRESSURES IN PATIENTS WITH NEUROLOGIC DISEASE RELATED TO CAROTID ARTERY LESIONS

Case No.	Diagnosis	Brachial Blood Pressures		Retinal Artery Pressures					
				Diastolic			Systolic		
		Right (mm. Hg)	Left (mm. Hg)	Right	Left	Diff. (percent)	Right	Left	Diff. (percent)
1	Thrombosis of R. Int. carotid	135/85	135/85	25	52	51.9	88	90	2.2
2	Thrombosis of L. Int. carotid	140/100	138/100	70	55	21.4	130	125	3.8
3	Thrombosis of L. Int. carotid	130/74	128/70	37	26	33.6	120	83	32.5
4	Thrombosis of L. Int. carotid	140/90	140/90	20	20	—	48	76	36.8
5	Ligation of L. carotid	134/82	126/86	25	25	0.0	118	78	33.9
	After compression of R. carotid			28	28	0.0	98	55	43.9
6	Ligation of L. common carotid			22	5	77.5	44	19	56.9
7	Ligation of L. Int. carotid	120/83	117/82	33	21	33.3	74	72	2.7
	After compression of R. carotid			33	20	39.4	70	30	57.1
8	Thrombosis of L. Int. carotid (Carcinoma in neck)	128/70	144/90	20	25	20.0	79	98	19.0
Range for 7 cases						0–77.5			2.2–57.1
Average % difference						31.7			24.1

TABLE 8

RETINAL-ARTERY PRESSURE FINDINGS IN TEN PREVIOUSLY REPORTED CASES WITH UNILATERAL INTERNAL CAROTID-ARTERY LESIONS

Author	Year	No. Cases	Retinal-Artery Pressures Equal	Ipsilateral Retinal-Artery Pressure Lower	Diagnosis
Schiff[21]	1935	1		1	Aplasia of left carotid with alexia
Krayenbuhl[20]	1944	2	2		Ligation of internl carotid after thrombosis
Miletti[22]	1946	1		1	Thrombosis of internal carotid
		4	1	3	Ligation of internal carotid
		2		2	Obstruction of internal carotid by tumor
		1		1	Obstruction of internal carotid by object
Total		11	3	8	

Comments

Fifteen of the total of 19 cases with carotid involvement and cerebral symptoms showed significant differences in retinal artery pressure of the two eyes. In all instances the difference far exceeded the range observed in the control series. Therefore, in only four cases in which cerebral symptoms were present, late collateral supply appeared sufficient to maintain approximately equal retinal artery pressures.

It is suggested by Sweet's[32] work (1948) that differences probably existed in the acute phase, as he had observed marked decrease of distal internal carotid pressure after proximal obstruction followed by gradual rising pressure as a result of increased collateral flow.

Miletti[22b] (1947) applied pressure over the carotid and observed a marked lowering of retinal artery pressure as in the case of ligation. He cites such an instance in which the retinal artery pressure fell to 0/0 initially and then rose after 12 hours to 20/40 gm., remaining at 25/40 gm. 14 days after ligation.

He has further clarified findings in cases of internal carotid involvement by reporting, in addition to marked differences between the retinal artery pressures on the two eyes, the following:

(1) Failure of carotid compression to alter the ipsilateral retinal pressure and (2) modification of the retinal artery pressure on the side with the lower systolic pressure by contralateral carotid artery compression.

Observations in three of our cases partially confirmed this type of finding. However, we were wary of routinely applying two minutes of compression to the single remaining patent carotid artery because of possible irreversible cerebral changes.

Actually, Miletti[22] found it necessary to desist in some cases because of "trouble de sensibilitiee. . . ." The occurrence of hemiplegia following routine carotid sinus compression should not be overlooked in this regard (Askey,[33] 1946; Zeman,[34] 1947).

Another objection to this additional measure of carotid compression lies in the doubt cast on its efficiency by the lack of consistent changes in directly measured internal carotid artery pressures after carotid artery compression through the skin (Sweet,[32] 1948).

In addition to variations in collateral flow related to time after occlusion, there are wide variations in anatomic structure of the collateral vessels (Eckstein,[35] 1941; Fetterman,[36] 1939; Govons,[37] 1946; Boyd,[38] 1934) as well as physiologic individual variation. These are suggested by the catastrophic effects of carotid-artery ligation in some individuals in contrast to the benign effects in others (Pilcher,[39] 1934; Dandy,[40] 1942; Shipley,[41] 1937; Fetterman,[36] 1939).

In an effort to select cases in which ligation is safe, Miletti[22b] (1947) suggests that, if carotid compression lowers the ipsilateral diastolic pressure less than 20 percent, ligation is safe, from 20 to 50 percent it is safe if done gradually, but unsafe if over 50 percent. This illustrates the range of variation found, but should be confirmed by clinical application and by direct observations such as Sweet's[32] (1948).

Most emphasis has been on systolic retinal-artery pressures, but our observations suggest that deviations in the diastolic pressures are significant as well.

The necessity for comparing bilateral brachial blood pressures to secure an adequate basis for comparison of retinal artery pressures is highlighted by the occurrence of lowered peripheral blood pressures on the side of the hemiplegia as illustrated in our Case 8 (Kerr,[28] 1936).

Other applications of this method offer attractive possibilities. Studies are projected or in process to evaluate the significance of single and serial retinal-artery pressures in the study of migraine, responses to various drugs in this and other conditions, and in the presence of tumors compared to acute cerebral thrombosis, hemorrhage, and so forth. In the latter condition, at least theo-

retical consideration suggests it might be useful in screening those cases in which sympathetic blocking techniques would be most successful.

A nontraumatic investigative technique such as this should be useful in differences in pressures of the two eyes in the presence of hemiplegia, aphasia, or convulsive disorders and could help differentiate those cases in which the primary disturbance is vascular.

Summary and Conclusions

Blood pressure in the retinal artery of each eye was measured in 58 cases by the method of Bailliart.[4] No significant inequality was found in 50 control cases or 199 reviewed controls. The average percent difference between the two eyes was 5.2 percent for the diastolic pressures and 3.3 percent for the systolic pressures.

Definite lowering of ipsilateral retinal-artery pressure was found in seven of eight cases with carotid artery lesions and in eight of 11 similar cases in the literature. Thus, the finding of equal retinal artery pressures does not exclude impairment of the internal carotid blood flow but, if other reasons for inequality are excluded, unilateral lowering of the retinal artery pressure is strongly suggestive evidence of interference with internal carotid blood flow.

In the presence of hemiparesis, aphasia, convulsive disorder, headache, or other evidence of cerebral dysfunction, a significant unilateral lowering of the retinal artery pressure is therefore suggestive of a pathologic condition of the internal carotid.

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References

1. Thomas, M. H.: Spontaneous carotid artery thrombosis. Thesis submitted for M.S. in Neurology, U. of Mich., Aug. 1, 1949 (to be published with additional cases, 1950).

2. Duke-Elder, W. S.: The ocular circulation: Its normal pressure relationships and their physiological significance. Brit. J. Ophth., **10**:513-572 (Oct.) 1926. Textbook of Ophthalmology. St. Louis, Mosby Company, v. 1, 1933.

3. Koch, F. L. P.: Ophthalmodynamometry. Arch. Ophth., **34**:234-247, 1945.

4. Bailliart, P.:
   (a) The Retinal Circulation (Translated by J. E. Lebensohn). Chicago, Professional Press, Inc., 1928.
   (b) L'hypertension arterielle retinienne. Internat. Cong. Ophth., **1**:87-142, 1938.
   (c) Quelques renseignements donnes par la mesure de la pression retinienne. Progres. Med., **2**: 21, 1947.

5. Espildora-Luque, C.: Ophthalmodynamometry and some of its clinical applications. Modern Trends in Ophthalmology (Sorsby). New York, Hoeber, 1947, v. 2.

6. Luo, T. H., and Ch'en, H. L.: Ophthalmodynamometric findings in the Chinese. Chinese, M. J., **56**:345-353, 1939.

7. Bardram, M., and Neerdam, H.: The value of ophthalmodynamometry ad modum Bailliart in intracranial tumors, Acta psychiat. and neurol., **19**:33, 1944.

8. Berens, C., Smith, H. T., and Cornwall, L. H.: Changes in the fundus and in the blood pressure in the retinal arteries in increased intracranial pressure. Arch. Neurol. & Psychiat., **20**:1151-1171 (Dec.) 1928.

9a. Uyemura, M., and Ishida, A.: Hydromechanic studies on the blood pressure of retinal blood vessels. Nippon Gankegakkai Zassi, **44**:1114, 1940. Cited in Far Eastern Science Bull., **1**:7 (May) 1941.

9b. Uyemura, M., and Suganuma, S.: Ueber einen neuen Ophthalmodynamometer. Acta Soc. ophth. jap., **38**:1835-1844, 1934.

10. Constantine, E. F.: A study of the interrelationship of retinal and systemic arterial pressure and intraocular tension in normal and syphilitic patients. Am. J. Ophth., **23**:436-445 (April) 1940.

11. de Sanctis, R.: Il comportamento della P.A.B. (Pressione arteriosa retinica) nelle diverse posizioni del corpo. Internat. Cong. Ophth. (1937), **2**:119-137, 1938.

12. Franceschetti, A., and Streiff, E. B.: Ueber die Beziehunger zwischen Netzhautarteriendruck und allgemeinem Blutdruck bei Diabetes. Klin. Monatsbl. f. Augenh., **102**:633-640, 1939.

13. Gutmann, A.: Pressure in central artery of retina in allergy with description of a new ophthalmodynamometer. Arch. Ophth., **29**:457-460 (Mar.) 1943.

14. Kapuscinski, W.: On symptoms of the pulse in the central retinal artery. Brit. J. Ophth., **32**: 881-885, 1948.

15. Lebensohn, J. E.: The determination of retinal blood pressure. Illinois M. J., **48**:212-214 (Sept.) 1925.

16a. Linksz, A., and Rasko, J.: Des relations entre la tension arterielle generale et retinienne et le tonus intraoculaire. Ann. d'ocul., **176**:747-751, 1939.

16b. Linksz, A.: Improved model of the Kukan ophthalmodynamometer. Am. J. Ophth., **25**:705-713 (June) 1942.

17. Magitot, A. P., and Bailliart, P.:

(a) Modifications de la tension oculaire sous l'influence de pressions exercees sur le globe (recherches experimentales). Ann. d'ocul., **156**:656-666 (Nov.) 1919.

(b) Le Pression vasculaire retienne dans un cas de cecite post-hemorrhagique. Ann. d'ocul., **156**: 666, 1919.

(c) How to know the blood pressure in the vessels of the retina. Am. J. Ophth., **5**:777-784 (Oct.) 1922.

18. de Morsier, G., Monnier, M., and Streiff, E. B.: (a) La tension arterielle retinienne dans les tumeurs intracraniennes. Revue neurol., **71**:702-714 (June) 1939.

(b) L'hypotension arterielle retinienne dans les tumeurs intracraniennes des etages anterieurs et moyens. J. de Belge neurol. et d psychiat., **39**:196, 1939.

19a. Streiff, E. B.: Le rapport entre la tension arterielle retinienne et la pression generale. Schweiz. med. Wchnschr., **67**:797-800, 1937.

19b. Tension arterielle retinienne. Tension oculaire sang. Ophthalmologica, **102**:104-119, 1941.

19c. Streiff, E. B., Montandon, A., and Monnier, M.: Variations simultanees des pressions arterielle retinienne et generale en jonction des excitations vestibulaires caloriques. Confinia neurologica, **4**: 347-350, 1942.

19d. Streiff, E. B., and Bischler, V.: The accuracy of ophthalmodynamometry. Ophthalmologica, **112**:19-24, 1946 (Quart. Rev. of Ophth., **4**:133, 1948).

20. Krayenbuhl, H., and Weber, G.: Die Thrombose der Arteria Carotis Interna und ihre Beziehung zur Endangiitis Obliterans, v. Winiwarter-Buerger. Helvet. med. acta, **11**:289-333, 1944.

21. Schiff-Wertheimer: In communication Laignier, M.: Bull. Soc. ophtal. Paris, March, 1935. Cited in Bailliart, P.: L'hypertension arterielle retinienne. Internat. Cong. Ophth., (1937), **2**:87-142, 1938.

22a. Miletti, M.: Diagnosis of primary thrombosis of internal carotid in cervical region by determination of values of systolic pressure of the central retinal artery. Presse med., **54**:655-656, 1946.

22b. Miletti, M., and di Luca, G.: Pressione arteriosa retinica e compressione della carotide comune: Sindrome tonometrica retinica da mancato circolo carotideo. Indice retinico di compenso cerebral e legatura della carotide comune (studio clinico sperimentale). Revista oto-neuro-oftal., **22**:1-31, 1947.

23. Friedenwald, J. S.: Retinal vascular dynamics. Am. J. Ophth., **17**:387-395, 1934.

24. Sobanski, J.: Ein Ophthalmodynamotonometer und seine Handhabung. Klin. Monatsbl. f. Augenh., **96**:351-356 (Mar.) 1936.

25. Kukan, F.: Ergebnisse der Blutdruckmessungen mit einem neuen Ophthalmodynamometer. Ztschr. f. Augenh., **90**:166-191 (Oct.) 1936.

26. Keil, J.: Ein neues Ophthalmodynamometer. Klin. Monatsbl. f. Augenh., **99**:625-636 (Dec.) 1937.

27. Bárány, E.: The influence of local arterial blood pressure on aqueous humor and intraocular pressure: An experimental study of the mechanisms maintaining intraocular pressure. Acta Ophth., **24**:337-387, 1946.

28. Kerr, W. J., and Underwood, F. J.: Hemiconstriction of the vascular system associated with cerebral disease. Am. Heart J., **12**:713-723, 1936.

29. Salvati, G.: La tension oculaire en position assise et couchee. Ann. d'ocul., **159**:128-130, 1922.

30. Baratta, O.: Studio sulla circolazione vascolare della retina con particolare riguardo al comportamento della pressione dell'arteria centrale (PAR) e dei capillari della macula (PCR) nelle affezioni renali. Ateneo parmense., **9**:265-295 (July-Aug.) 1937.

31. Sabbadini, D.: La ipertensione autonoma minimo comune desspressionatore di varie gravi complicanze oculari. Internat. Cong. Ophth. (1937), **2**:56, 1938.

32. Sweet, W. H., and Bennett, H. S.: Changes in internal carotid pressure during carotid and jugular occlusion and their clinical significance. J. Neurosurg., **5**:178, 1948.

33. Asky, J. M.: Hemiplegia following carotid sinus stimulation. Am. Heart J., **31**:131-137, 1946.

34. Zeman, F. D., and Siegal, S.: Monoplegia following carotid sinus pressure in aged. Am. J. M. Sc., **213**:603-607, 1947.

35. Eckstein, P. W., Gregg, D. E., and Pritchard, W. H.: The magnitude and time of development of collateral circulation in occluded femoral, carotid and coronary arteries. Am. J. Physiol., **132**:351-361, 1941.

36. Fetterman, J. L., and Pritchard, W. H.: Cerebral complications following ligation of the carotid artery. J.A.M.A., **112**:1317-1322, 1939.

37. Govons, S. R., and Grant, F. C.: Arteriographic visualization of cerebrovascular lesions. Arch. Neurol. & Psychiat., **55**:600-618, 1946.

38. Boyd, J. D.: Absence of the right common carotid artery, J. Anat., **68**:551-557, 1934.
39. Pilcher, C., and Thuss, C.: Cerebral blood flow: Cerebral effects of occlusion of the common or internal carotid arteries. Arch. Surg., **29**:1924-2038, 1934.
40. Dandy, W. E.: Results following ligation of internal carotid artery. Arch. Surg., **45**:521-533, 1942.
41. Shipley, A. M., Winslow, N., and Walter, W. W.: Aneurysm in the cervical portion of the internal carotid artery. Ann. Surg., **105**:673-699, 1937.

CENTRAL RETINAL ARTERY OCCLUSION IN ORBITAL INFLAMMATION*

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AND

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Since Hippocrates reported his patient, Hegesistratius,[1] who had suppuration near the eye and thick matter in the nostrils, orbital cellulitis and abscess formation has been recognized as a complication of sinus infection. The purpose of this paper is to report a case of secondary orbital infection, peculiarly complicated by occlusion of the central retinal artery and to analyze the mechanisms involved.

CASE REPORT

A 56-year-old white woman was admitted to Dr. Shipman's service (Hosp. No. 4076), Wills Eye Hospital, August 29, 1950, complaining of a painful swelling about the right eye which had developed during the previous night. She felt weak and lethargic that night and on awakening found her right lids swollen and adherent.

For two months she had noted a painless, subcutaneous lump, approximately one cm. in size, appearing about twice a week above the right, medial canthal ligament. She could reduce this mass by digital pressure; simultaneously, yellowish fluid would appear in her right nostril.

Past history was negative except for a chronic postnasal discharge and a tendency toward more profuse rhinorrhea on the right in association with colds during the previous four months. She did not have previous headaches, visual disturbances, or eye disorders.

Examination revealed an obese woman in marked discomfort. Temperature was 102°F. Vision and detailed study of the left eye and adnexa were normal.

Vision in the affected right eye was 6/9—4, unimprovable with a pinhole. The right lids were hyperemic and showed three-plus, nontender edema closely limited to the orbital area. A tender, fluctuant, subcutaneous mass, 1.5 cm. in diameter, was present above the medial canthal ligament.

On forced opening of the right lids there was immediate diplopia. The right globe was proptosed five mm., and ocular rotations were restricted in all directions. Moderate chemosis was present. The pupils were round and equal, reacting properly to light and to convergence.

Slitlamp study of the anterior segment was negative. Ophthalmoscopic examination revealed a violent, arterial pulsation with complete blanching of the nervehead during cardiac diastole. There were no other fundus abnormalities. Intraocular pressure was 89 mm. Hg (Schiøtz).

Laboratory. Admission studies were reported as follows:

a. White blood count 10,000; neutrophilic granulocytes 78 percent; lymphocytes 20 percent.

b. Red blood count 4,210,000; hemoglobin 74 percent.

c. Routine blood sugar and urinalysis: normal.

d. X-ray studies of paranasal sinuses: dense cloudiness in right antrum and ethmoid cells without evidence of bone involvement (fig. 1).

e. Diagnostic irrigation of right antrum produced one ounce of foul yellow-green pus.

f. Intranasal cultures: reported as hemolytic Staphylococcus albus and Bacillus pyocyaneus.

g. Blood Kahn, 4+; blood Wassermann, 4431.

The patient was placed on hot compresses and depopenicillin (300,000 units, twice daily). Temperature the next day dropped slightly but orbital edema was worse and the globe was immobile. Streptomycin (0.5 gm., twice daily) and sulfa-

* From the Wills Eye Hospital. Presented at the monthly staff conference, October, 1950.

diazine (4.0 gm., immediately, and 1.0 gm., every six hours, covered by $NaHCO_3$), were added to the treatment. A repeat antral irrigation produced a "dishwater" return.

On the third hospital day temperature dropped to 100°F. but the proptosis and lid edema were worse. Pyribenzamine (50 mg., every six hours) was ordered to aid nasal drainage because the appearance of the nasal mucosa and a six-percent eosinophilia suggested the presence of complicating allergy.

The following morning (September 1st) vision in the right eye was reduced to poor light perception. Fundus examination was difficult, due to lid edema, but revealed an occlusion of the central retinal artery. On this fourth day, a right intranasal ethmoidectomy and a naso-antral fenestration were done. The fluctuant mass above the inner canthal ligament drained through the ethmoidectomy site, but within the ethmoidal cells there was no free-flowing pus or angry edema.

On the fifth day (September 2nd) the patient's temperature settled below 100°F. but swelling was still intense and chloramphenicol* (3.0 gm., immediately, and 0.5 gm., every six hours) was added to the therapeutic program.

Fundus examination revealed extremely attenuated retinal arterioles and a cherry-red spot at the macula in the midst of diffuse pale edema of the posterior pole. No hemorrhages or exudates were present. The orbit and fundus remained unchanged through the sixth day, but all traces of light perception faded.

Edema and discomfort began to subside at the end of the first week, but another abscess localized below the right supraorbital notch, requiring incision on the eighth day. White blood count at this time was 12,050 but polys had dropped to 63 percent and lymphocytes increased to 30 percent. Creamy yellow pus was delivered from a localized abscess cavity and a drain was inserted. There was little subsequent drainage.

During the next 10 days there was slow recession of the orbital swelling and intraocular hypertension. Ocular mobility began to return. Repeat X-ray studies (September 7th) showed no appreciable change, which is in accord with the usual persistence of roentgen findings after clinical improvement. White blood count dropped to 5,300 on September 9th and sulfadiazine was discontinued. Chloramphenicol was continued until September 13th.

On the 19th day (September 16th), a third abscess localized above the site of the original mass near the inner canthus. This was incised and a drain passed through the skin into the nasal cavity via the exenterated ethmoidal area. Again there was little subsequent drainage. White blood count at this time was 8,700.

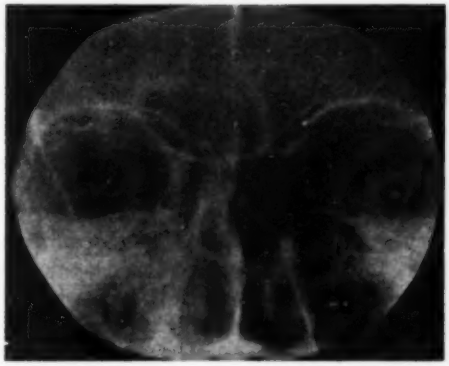

Fig. 1 (Keeney, Kasper, and Shipman). Roentgen appearance of orbits and paranasal sinuses at time of admission.

Repeated blood Kahn and Wassermann tests confirmed the admission serology but spinal-fluid studies were negative. By the end of September, ocular rotations were normal but 1.5 mm. of proptosis persisted. There has been no restoration of vision and the nervehead became dull white with a deep central cup. X-ray studies on the 36th day showed moderate clearing in the antrum and ethmoidal areas with no evidence of osteomyelitis.

A recurrence of fluctuation at the original site above the medial canthal ligament required incision on the 39th day. Cultures from this area yielded Proteus mirabilis.† Streptomycin, which had been discontinued after a 14-day course, was resumed.

Studies for blood sludging[3] on the 46th day revealed marked evidence of sludged blood in the conjunctival vessels of both eyes. Such findings, however, have been reported in apparently normal individuals[3] and are of questionable diagnostic value.

Close observation for one year has revealed no further evidence of inflammatory activity, though some increased retrobulbar resistance remained in the right orbit for several months.

Comment

The basic diagnosis of fulminating orbital inflammation secondary to sinusitis is clear in this case, but several differential diagnoses should always be considered at the initial examination of such patients. These are (a)

* Subsequent studies on the antagonism of chloramphenicol to penicillin suggest that this was probably an unwise combination of antibiotics (Jawetz, E., Gunneson, J., Speck, R., and Coleman, V.: Interference of chloramphenicol with penicillin. Arch. Int. Med., 87:349 (Mar.) 1951).

† Proteus organisms, generally nonsensitive to penicillin, may be expected to appear in overgrowth and assume pathogenicity due to the long course of penicillin (Leopold, I. H., and LaMotte, W. O.: Influence of penicillin on the course of ocular lesions due to a toxic agent. Am. J. Ophth., 30:41 (Jan.) 1947).

collateral edema secondary to adjacent sinus infection, (b) serous tenonitis, (c) malignant anthrax edema without pustule, (d) cavernous sinus thrombosis, and even (e) frontal lobe abscess.

Mechanisms of arterial occlusion

Serious complications of orbital infection such as malignant exophthalmos, cavernous sinus thrombosis, meningitis, and death are familiar to all who recall the days before sulfa drugs. Visual complications from optic-nerve affections are often mentioned but their pathogenesis has not been clear.

Central retinal-artery occlusion as a complication of orbital infection is not mentioned in standard texts, or in studies of etiology in arterial occlusion. In recent literature, only three case reports[4-6] have been found and two of these attribute the occlusion to mechanical pressure of the swollen orbital tissue—a process, if so simple and direct, which might logically be expected to obstruct the thinner walled, central retinal vein, before compressing the tougher artery. The third report[4] reiterates the early opinion of Arnold Knapp* (1906) that such catastrophies may be due to arteritis.

Possible mechanisms in such an occlusion are:

1. *Direct mechanical pressure.* This would necessitate a site of action posterior to points where the central vessels are together in order to avoid venous occlusion prior to arterial occlusion. Both the extraneural and intraneural course of these vessels are quite variable.[7]

The vein is commonly subject to more acute angulations than the artery. There is no specific anatomic situation rendering the artery more vulnerable than the vein to increased intraorbital pressure. Potentially, triple drainage of the vein through both ophthalmic veins and the angular vein increases, however, the area over which pressure would have to be exerted in order to occlude in the extraneural course the vein as compared to the artery.

Normal intra-arterial pressure (approximately 95 mm. Hg, systolic) being more than four times as great as the usual intravenous pressure (less than 20 mm. Hg) would still seem to render the venous circulation subject to earlier obstruction if the mechanism were by direct pressure.

Stretching of the nerve has been postulated to cause arterial occlusion, but this is primarily a variant of the pressure theory and would require an exophthalmos greater than 7.0 mm. to take up the usual slack in the optic nerve.

2. *Spasm* due to local "toxins" may be responsible. Occurrence of angiospasm in this vessel has been well verified, and the vein, because of its different histology and innervation, is probably unable to go into spasm.

3. *Increased intraocular pressure* per se can prevent the access of blood to the eye[8] and, in this case, the tension was very high at the time of occlusion. Such glaucoma results from (a) splinting of the globe by swollen orbital contents or (b) mechanical occlusion of vortex or ciliary veins.

Glaucoma from vortex or ciliary vein occlusion would be free of retinal hemorrhages as seen in central retinal-vein obstruction but such mechanism is eliminated in this case by the brief duration of hypertension.

Glaucoma from experimental ligation[9] or accidental electrocoagulation of a vortex vein (as during retinopexy) usually persists for a few weeks at least. Glaucoma following experimental ligation of anterior ciliary veins lasts for months.

4. *Blood sludge* (Knisley, and others) due to locally elaborated sludging factors in the inflamed area is probably a factor in cases such as this. On the basis of lumen diameter, a given amount of sludge would be able to occlude the smaller artery more easily than the larger vein.

* Knapp, A. (Arch. Ophth., 35:524 (June) 1906) has presented three similar cases followed by blindness, but the published evidence to justify including them here is not clear.

The direction of movement of intra-arterial sludge would be more likely to produce fundus symptoms than would intravenous sludge which tends to flow away from the globe. In the presence of sludge, treatment with large amounts of heparin may facilitate circulation.

5. *Septic endarteritis* by direct invasion of the vessel wall may obstruct the blood flow. The arterial lumen is about 0.13 mm. in diameter, whereas the venous lumen is nearly twice that diameter. Even in simultaneous infection of both vessels, the smaller artery might be occluded first.

As a consequence of such spread of contiguous infection, septic or necrotic emboli may destroy vision.

Discussion

Although autopsy verification is lacking, the mechanism in this case apparently was that the elevated intraocular pressure was greater than the systolic pressure in the central retinal artery. This precluded the arterial blood supply within the globe; retinal hemorrhages, which would have accompanied an antecedent venous occlusion, were lacking. The secondary glaucoma was due to splinting of the globe by swollen orbital tissues.

Multiple mechanisms, such as associated blood sludge or septic endarteritis, may have had a part but the simple explanation of orbital pressure acting directly and selectively on the central retinal artery is not tenable.

In this type of case, immediate and intensive therapy must be directed to controlling the basic inflammatory disease; however, the intraocular pressure should also be carefully followed. If the tension approaches average systolic pressure in the central retinal artery, as indicated by Schiøtz readings of between 80 and 90 mm. Hg, or by complete blanching of the nervehead during cardiac diastole, orbital decompression by the subzygomatic route is indicated.

The Kroenlein external orbitotomy, the Shagrue operation, or the Guyton technique are procedures well suited to this condition and should be considered as sight-saving emergency measures. When the threat to life may adequately be met with antibiotics, and such surgery for sight, with its attendant general anesthetic risk, is contraindicated, the early use of heparin should be seriously considered.

Summary

A case of fulminating orbital inflammation secondary to sinus infection has been presented and the differential diagnosis outlined. An unusual complication—central retinal-artery occlusion—occurred. The mechanisms involved in such occlusions have been outlined. These may occur singly or in combination and indicate corresponding medical and surgical therapies which may require prompt administration for both sight and life.

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References

1. Littre, M. P. E.: Oeuvres completes d'Hippocrate. Paris, J. B. Bailliere, Tome Cinquiene, 1846, p. 169.
2. Knisley, M., and Bloch, E.: Intravascular agglutination of red cells and blood sludge in human disease. Anat. Rec., **82**:426 (Mar. supple.) 1942.
3. Robertson, H., Wolf, S., and Wolff, H.: Blood sludge in human subjects. Am. J. Med. Sc., **219**: 534 (May) 1950.
4. Green, J.: Orbital cellulitis. Am. J. Ophth., **9**:34 (Jan.) 1926.
5. Ballou, O. E.: Orbital cellulitis followed by blindness. Memphis M. J., **22**:184 (Nov.) 1947.
6. Donahue, H. C.: Orbital cellulitis followed by blindness. Am. J. Ophth., **29**:1574 (Dec.) 1946.
7. Fry, W. E.: Variations in the intraneural course of the central vein of the retina. Arch. Ophth., **4**:180 (Aug.) 1930.
8. Adler, F. H.: Physiology of the Eye. St. Louis, Mosby, 1950, p. 121.
9. Duke-Elder, W. S.: Textbook of Ophthalmology. St. Louis, Mosby, 1940, v. 3, p. 3326.

THE VON GRAEFE AND KERATOME INCISION IN CATARACT EXTRACTIONS*

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The purpose of this paper is to compare the operative technique, postoperative course, and visual results of two series of cataract extractions. In one series the corneal section was made with the classical Graefe or Smith-Green knife, and in the second series a keratome was used, the section being enlarged with scissors. There was a total of 150 cases in each series. Six surgeons, three in each series, performed all the operations.

All of the cases were operated on by members of the Wilmer Institute staff. With the exception of 50 keratome cases, all operations were done in the Wilmer Institute while the operator held the position of resident surgeon. All the cases chosen for study were consecutive, uncomplicated senile cataracts, either immature, mature, or hypermature and, insofar as could be preoperatively determined, were not associated with any intraocular diseases.

Each was a simple, round pupil, intracapsular extraction with peripheral iridotomy. In every case, the pupil was routinely dilated with five-percent homatropine prior to operation, occasionally augmented by 10-percent neosynephrine. The routine was a retrobulbar injection of two- or four-percent procaine hydrochloride combined with either a Van Lint or a seventh nerve block. In some cases sodium pentothal was used.

In every case, a superior rectus suture was employed and two 6-0 McLean-type corneoscleral sutures were used. The cross-action Castroviejo intracapsular forceps were routinely used, except in a few cases in which the Arruga forceps or Bell erisophake was employed.

Following delivery, and after tying the sutures, a miotic was instilled. For the most part, only the operated eye was bandaged.

The patient was elevated 45 degrees on the day of operation. On the day following operation, the eye was dressed, the pupil dilated with atropine, and the patient allowed to get out of bed. The eye was subsequently dressed daily with atropine.

The corneoscleral sutures were usually removed about the 10th day. In uncoöperative patients, the sutures were allowed to remain in longer or permitted even to slough out.

In most cases, upon leaving the hospital, a temporary aphakic correction was given each patient. The final refraction was not done until at least six weeks following operation.

With the exception of the type of incision, this same routine procedure was followed in every case.

Two of the surgeons using the keratome technique entered the anterior chamber at the 12-o'clock position between the two corneoscleral sutures; the other surgeon passed the instrument between one of the sutures. The incision was enlarged with scissors to include approximately the upper half of the cornea. The surgeons who used the Graefe knife made an incision which included at least the upper two thirds of the cornea and, when necessary, extended the section with scissors to include the upper half of the cornea.

This series is broken down into four parts: (1) The operative complications, (2) the postoperative course, (3) the immediate postoperative complications, and (4) the final visual result.

The operative complications specifically noted were whether or not the lens capsule was broken during delivery and whether there was any vitreous lost. In the post-

* From the Wilmer Ophthalmological Institute of the Johns Hopkins Hospital and University.

operative course, the complications recorded were the degree of conjunctival reaction, the amount of striate keratitis, and any delayed reformation of the anterior chamber. The evaluation of the first two depended upon the opinion of the observer and were graded one, two, and three plus.

Results

Table 1 shows the operative complications. Using the keratome, the capsule was broken in 26 or 17.3 percent; with the Graefe knife, 25 capsules or 16.6 percent were broken.

The question of vitreous loss is difficult to evaluate. In the Wilmer Institute, the operative note is dictated immediately at the close of operation. If there is the slightest question of vitreous loss, even of fluid vitreous, the case is recorded as "vitreous loss." The degree of loss is qualified "a drop of vitreous escaped through the wound" or "a strand of vitreous followed the lens," "slight loss," "moderate loss," and so forth.

The incidence of vitreous loss here reported includes all cases questionable and definite, irrespective of the degree. For the sake of simplicity, vitreous loss in the data just given is listed as Grades 1, 2, and 3.

With the keratome incision, there was vitreous loss in 12 cases or 8.0 percent and when the Graefe knife was used, 14 cases or 9.2 percent.

Table 2 illustrates the postoperative course. There appeared to be less conjunctival reaction with the Graefe-knife incision than with the keratome incision. About the same amount of striate keratitis was present in both series. The anterior chamber reformed about the same time with each incision.

Table 3 illustrates the postoperative complications.

Anterior-chamber hemorrhages occurred in 27 or 18 percent of those cases in which the keratome was used, and 18 or 12 percent of those cases in which the Graefe knife was used. This possibly significant difference perhaps is due to the fact that when the keratome is used to make the initial incision, the incision may be more scleral and the en-

TABLE 1

Operative complications

	Broken Capsule	Vitreous Loss 1	Vitreous Loss 2	Vitreous Loss 3	Total
Keratome-scissors Incision (150 cases)	26 or 17.3%	5 or 3.3%	5 or 3.3%	2 or 1.3%	12 or 8.0%
Graefe-knife Incision (150 cases)	25 or 16.6%	8 or 5.3%	2 or 1.3%	4 or 2.6%	14 or 9.2 %

TABLE 2

Postoperative course

	Conjunctival Reaction			Striate Keratitis			Date Anterior Chamber Reformed			
	1	2	3	1	2	3	1	2	3	3+
Keratome-scissors Incision (150 cases)	115 or 76.6%	29 or 19.3%	5 or 3.3%	102 or 82.9% (123 eyes evaluated)	19 or 15.4%	2 or 1.6%	92 or 94.8% (97 eyes evaluated)	1 or 1.3%	3 or 3.0%	1 or 1.3%
Graefe-knife Incision (150 cases)	133 or 88.6%	15 or 10.0%	2 or 1.3%	89 or 80.1% (111 eyes evaluated)	17 or 15.4%	5 or 4.5%	93 or 93% (100 eyes evaluated)	2 or 2%	2 or 2%	3 or 3%

TABLE 3

POSTOPERATIVE COMPLICATIONS

	Anterior Chamber Hemorrhage	Iris Prolapse	Delayed Reforming of Anterior Chamber
Keratome-scissors Incision (150 cases)	27 or 18.0%	5 or 3.3%	16 or 10.6%
Graefe-knife Incision (150 cases)	18 or 12.0%	4 or 2.6%	16 or 10.6%

largement of the wound with scissors may be somewhat deeper.

Iris prolapse occurred in five or 3.3 percent of those cases in which the keratome was used and four or 2.6 percent of those in which the Graefe knife was used.

Delayed reformation of the anterior chamber occurred in equal numbers, being 16 or 10.6 percent in each series.

Table 4 shows the final vision.

With the keratome incision 99 or 66 percent had 20/20 vision; 33 or 22 percent had 20/30 vision; 11 or 7.3 percent had 20/40 vision; four or 2.6 percent had 20/50 vision; one patient had 20/70 vision, and two had 20/100 vision.

With the Graefe knife incision, 98 or 65.3 percent had 20/20 vision; 34 or 22.6 percent had 20/30 vision; 12 or 8.0 percent had 20/40 vision; three or 2.0 percent had 20/50 vision; one had 20/70 vision, and two had 20/100 vision. In short, the visual results were consistently identical in each series.

TABLE 4

FINAL VISION

Final Vision	Keratome-Scissors Incision (150 cases)	Graefe-Knife Incision (150 cases)
20/20	99 or 66.0%	98 or 65.3%
20/30	33 or 22.0%	34 or 22.6%
20/40	11 or 7.3%	12 or 8.0%
20/50	4 or 2.6%	3 or 2.0%
20/70	1 or 0.6%	1 or 0.6%
20/100 and less	2 or 1.3%	2 or 1.3%

Table 5 shows the residual astigmatism in each series.

There was no astigmatism in 27 or 18 percent of the keratome incision cases and in 22 or 14.6 percent of the Graefe knife incision cases. With the keratome incision, less than 1.0D. was found in 38 or 25.3 percent; 1.0D. to 1.75D. in 38 or 25.3 percent; 2.0D. to 2.75D. in 34 or 22.6 percent and 3.0D. and over in 13 or 8.6 percent of the cases.

TABLE 5

ASTIGMATISM

	Keratome-Scissors Incision (150 cases)	Graefe-Knife Incision (150 cases)
No astigmatism	27 or 18.0%	22 or 14.6%
Less than 1.00 diopter	38 or 25.3%	34 or 22.6%
1.00 to 1.75 diopters	38 or 25.3%	50 or 33.3%
2.00 to 2.75 diopters	34 or 22.6%	34 or 22.6%
3.00 diopters and over	13 or 8.6%	10 or 6.6%

TABLE 6

VITREOUS LOSS

Keratome-scissors Incision (150 cases)	20/20 —4 20/30 —4 20/40 —2 20/50 —1 20/100—1
Graefe-knife Incision (150 cases)	20/20 —9 20/30 —2 20/40 —3

With the Graefe knife incision, less than 1.0D was found in 34 or 22.6 percent; 1.0D. to 1.75D. in 50 or 33.3 percent; 2.0D. to 2.75D. in 34 or 22.6 percent; and 3.0D. and over in 10 or 6.6 percent of the cases.

Again there was no significant statistical difference in the two series.

Table 6 shows the final visual result in those cases in which there was vitreous loss. Peculiarly enough, there was no significant difference in the final result of the cases in which vitreous was lost when compared to those in which there was no loss.

Comment

Except for the slightly more frequent occurrence of anterior-chamber hemorrhage when the keratome incision was used—18 percent versus 12 percent—there was no statistical difference in the operation complications, postoperative course, and final vision in the two series. However, in spite of this statistical similarity, it is our general impression that there was a slightly greater postoperative reaction in the cases in which the keratome was used to make the section.

This study gives no information which would support one or the other types of corneal section. Possibly analysis of a larger series of cases might yield some indication that one method was preferable to the other. This series, however, is sufficiently large to indicate that there is little, if any, difference in the over-all course or the final visual result following these two techniques.

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NEW AIR FORCE VISUAL AND OPHTHALMOLOGICAL REQUIREMENTS*

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Recently a new regulation† was published covering medical requirements for the various flying and nonflying personnel in the U. S. Air Force. The eye requirements are considered of such interest to ophthalmologists that they have been tabulated and are presented herewith (table 1).

In addition, tables are enclosed showing minimal accommodative power for age (table 2), and normal mean accommodative power values for age (table 3).

Causes for rejection for the various ophthalmological conditions are listed as follows:

1. Nonacceptable for enlistment

a. *Lids:* (1) Trichiasis; (2) complete or extensive destruction of the lids sufficient to impair protection of the eye from exposure; (3) disfiguring cicatrices and adhesions of the lids to each other or to the eyeball; (4) chronic severe blepharitis; (5) ptosis interfering with vision; (6) entropion, ectropion, or lagophthalmos; (7) malignant growths; (8) acute or chronic dacryocystitis.

b. *Conjunctiva:* (1) Acute or chronic conjunctivitis, including vernal catarrh, if more than mild; (2) trachoma, active.

c. *Cornea:* (1) Acute or chronic keratitis; (2) intractable or recurrent ulcers.

d. *Uveal tract, retina, and optic nerve:* (1) acute, chronic, or recurrent inflammation of the uveal tract; (2) retinitis; (3) neuroretinitis; (4) optic neuritis; (5) papilledema; (6) bilateral optic atrophy; (7) pigmentary degeneration of the retina.

e. *Lens:* (1) Opacities of the lens, presumably progressive; (2) dislocations of a lens.

f. *Diseases of the eye:* (1) Primary glaucoma; (2) secondary glaucoma; (3) night blindness, due to objective organic disease of the eye. (4) malignant tumor; (5) exophthalmos sufficient to interfere with proper closure of the lids and protection of the cornea; (6) diplopia, if uncorrected; (7) progressive tumors of the orbit and/or disturbance of vision or impairment of visual fields due to disease of the brain; (8) loss of one eye or anophthalmos; (9) any organic disease of the eye or adnexa not already specified, which threatens continuity of visual fields or impairment of visual function; (10) a heterotropia of more than 15 degrees.

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* From the office of the Surgeon General, United States Air Force.

† Refers to AFM 160-1, 1 September 1951, U. S. Government Printing Office, Washington, D.C., and changes.

TABLE 1

EYE REQUIREMENTS—U. S. AIR FORCE

	Distant Vision	Near Vision	Refractive Error	Motility	Accommodation	Color Vision	Depth Perception	Visual Fields	Night Vision*	Red Lens Test
Flying Training (Pilot) Class I	20/20 O.U.	20/20 O.U.	Total +1.75 −0.25 Astig. ±0.75	Eso 10 Exo 5 Hyper 1 Pc 70 or Less	At least minimum for age (table 2)	Must make 4 or less errors on 17 Plate AOC	Passing Score—(Verhoeff) or 30 (Howard-Dolman) or No errors—(Armed Forces Vision Tester)	15° Loss in any Meridian. Scotoma disqualifies	Satisfactory score SAM Tester	No diplopia within 20° of center of screen in cardinal directions
Flying Training (Observer) Class IA	20/50 O.U. Correctible to 20/20 O.U.	Same as Class I	X	Same as Class I	At least mean for age (table 3)	Same as Class I	Same as Class I	Same as Class I	Same as Class I	Same as Class I
Flying (Training Completed) Class II	Same as Class IA	20/50 O.U. Correctible to 20/20 O.U.	X	Eso 10 Exo 5 Hyper 1.5 Pc 70 or Less	Same as Class I	Must make 4 or less errors on 17 Plate AOC unless makes score of 50 or better on SAM CTT	Same as Class I	15° Loss or Scotoma due to active process disqualifies	Same as Class I	Same as Class I
Flying (For personnel not in primary control of aircraft) Class III	20/200 O.U. Correctible to 20/20 and 20/30	Correctible to 20/20 and 20/30	X	X	X	Same as Class II	X	Same as Class II	Same as Class I	X
Commission (Nonflying)	Same as Class III	Correctible to 20/20 and 20/40	Total +5.00 −3.00	Eso 15 Exo 8 Hyper 2	X	X	X	Defects which interfere significantly w/binocular vision disqualify	Night Blindness disqualifies	X
Control Tower Operator	Correctible to 20/20 O.U.	Correctible to 20/20 O.U.	X	Eso 10 Exo 5 Hyper 1.5	Same as Class II	Same as Class II	Same as Class II	Same as Class II	Same as Class II	Same as Class II
Radar Operator	X	Correctible to 20/20 O.U.	X	Eso 10 Exo 5 Hyper 1	Same as Class IA	X	X	X	Same as Class I	X
Marine Diving Duty	20/30 O.U. Without Correction	X	X	X	X	Same as Class I	X	X	X	X
Enlistment	Correctible to 20/40 and 20/70 or 20/30 and 20/100 or 20/20 and 20/400 or less, if not due to progressive disease	X	X	X	X	X	X	X	X	X

* = Not required unless history shows evidence of defective night vision.
X = No standards.

2. NONACCEPTABLE FOR COMMISSION

a. *Lids:* (1) Trichiasis; (2) destruction of the lids sufficient to impair protection of the eye from exposure; (3) disfiguring cicatrices and adhesions of the lids to each other or to the eyeball; (4) blepharitis, chronic, unless it is the opinion of the examiner that it is sufficiently mild in degree to interfere in no way with performance of duty; (5) ptosis interfering with vision; (6) entropion or ectropion; (7) lagophthalmos; (8) growth or tumor of the eyelid other than asymptomatic, nonprogressive small benign lesions; (9) dacryocystitis, acute or chronic; (10) epiphora.

b. *Conjunctiva:* (1) Conjunctivitis, acute, until recovered; (2) conjunctivitis, chronic, including vernal catarrh; (3) trachoma, unless healed without cicatrices; (4) xerophthalmia; (5) pterygium which encroaches on the cornea more than one millimeter or is progressive, as evidenced by marked vascularity or a thick, elevated head.

c. *Cornea:* (1) Keratitis, acute or chronic; (2) corneal ulcer or history of recurrent ulcers.

d. *Uveal tract, retina, and optic nerve:* (1) Inflammation of the uveal tract (iris, ciliary body, or choroid), acute, chronic, or recurrent; (2) neuroretinitis; (3) choroidoretinitis, unless healed, considered unlikely to recur and not interfering significantly with visual function; (4) optic neuritis; (5) papilledema; (6) optic atrophy; (7) pigmentary degeneration of the retina; (8) coloboma of the choroid or iris.

e. *Lens:* (1) Opacities of the lens which are considered to be progressive or which interfere in any way with vision; (2) dislocation of a lens.

f. *Diseases of the eye:* (1) Glaucoma, primary or secondary; (2) tumor of the eye; (3) anophthalmos; (4) exophthalmos; (5) night blindness; (6) asthenopia; (7) entropion or ectropion; (8) lagophthalmos or dacryocystitis.

g. *Ocular mobility:* (1) Nystagmus; (2) heterotropia; (3) diplopia.

TABLE 2

ACCOMMODATIVE POWER—MINIMUM FOR AGE

Age (years)	Diopters	Age (years)	Diopters
17	8.8	31	5.4
18	8.6	32	5.1
19	8.4	33	4.9
20	8.1	34	4.6
21	7.9	35	4.3
22	7.7	36	4.0
23	7.5	37	3.7
24	7.2	38	3.4
25	6.9	39	3.1
26	6.7	40	2.8
27	6.5	41	2.4
28	6.2	42	2.0
29	6.0	43	1.5
30	5.7	44	1.0
		45	.6

TABLE 3

ACCOMMODATIVE POWER—NORMAL MEAN VALUE OF AGE

Age (years)	Diopters	Age (years)	Diopters
17	11.8	31	8.4
18	11.6	32	8.1
19	11.4	33	7.9
20	11.1	34	7.6
21	10.9	35	7.3
22	10.7	36	7.0
23	10.5	37	6.7
24	10.2	38	6.4
25	9.9	39	6.1
26	9.7	40	5.8
27	9.5	41	5.4
28	9.2	42	5.0
29	9.0	43	4.5
30	8.7	44	4.0
		45	3.6

h. *Other organic diseases of the eye or adnexa:* Any organic disease of the eye or adnexa not already specified, which threatens continuity of vision or impairment of visual function.

3. NONACCEPTABLE FOR FLYING—CLASSES I AND IA

a. History of choroidoretinitis.

b. Any organic disease of the eye or adnexa not already specified, which threatens continuity of vision or impairment of visual function and, in the opinion of the examiner, will in any way interfere with efficient performance of flying duty or the individual's well being.

4. NONACCEPTABLE FOR FLYING—CLASSES II AND III

a. Any organic disease of the eye or adnexa not already specified, which threatens continuity of vision or impairment of visual function and, in the opinion of the examiner, will in any way interfere with efficient performance of flying duty or the individual's well being.

b. The disqualifying conditions listed under paragraphs 2 and 3, when found in rated and trained personnel, will be individually evaluated in regard to severity, prognosis, and importance in relationship to continued flying.

SUMMARY

New visual and ophthalmological requirements for the various classes of examinations used in the U. S. Air Force, as prescribed in Air Force Manual (AFM) 160-1, are presented.

Headquarters U. S. Air Force (25).

EVISCERATION WITH PLASTIC INTRASCLERAL IMPLANTS*

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Since Mules,[1] in 1884, devised a hollow spherical glass implant, no radical changes in the technique of evisceration were proposed up to 15 years ago, except that a cylindric implant was substituted for the sphere.

Evisceration has gained more popularity in European countries than in the United States, where the preference is for enucleation. The preference for enucleation where evisceration is indicated is difficult to comprehend in view of the fact that the technique is simple, has proven to be safe in a large number of cases, and there is less danger of extrusion.

Psychologically, a patient more readily accepts the removal of the contents of the eye rather than the removal of the globe. The cosmetic results are good, usually with less sinking of the upper eyelid, and the movement of the prosthesis more normal because the muscles are not disturbed.

Excising the optic nerve during enucleation produces a potential pathway for infection by way of the meninges of the optic nerve to the meninges of the brain. Evisceration has been repeatedly advocated in cases of panophthalmitis[2] to prevent meningitis. Thrombosis of the cavernous sinus has been frequently observed in reported cases of meningitis following enucleation, but it has not been determined clinically whether meningitis was the result of bacteria invading the optic-nerve sheaths or due to surgical trauma.

Extensive research work by British ophthalmologists[3] and others[4] indicates that sympathetic ophthalmia does not follow evisceration more often than enucleation. However, if an eye is potentially sympathicogenic, neither evisceration nor enucleation will always prevent sympathetic ophthalmia.

The high incidence of extrusion of implants following the Mules operation reported by Verrey[5] and Burch[6] may have been due to the fact that (1) the sphere was too large to be retained in the scleral shell, (2) hemorrhage had not been controlled, or (3) the sclera was not adequately closed with nonabsorbable sutures.

In view of the poor results obtained with various types of integrated implants, it was considered that a report of experience with

* From the Department of Research, New York Eye and Ear Infirmary, and the Department of Ophthalmology, New York University Postgraduate Medical School. Aided by grants from the Ophthalmological Foundation, Inc., and the Snyder Foundation.

TABLE 1

EXTRUSION OF IMPLANTS FOLLOWING EVISCERATION

Reported By	Type of Implant	Total No. of Cases	Extrusion (%)
Verrey	(Hollow glass spheres Mules)	343	21.3
Burch	(Hollow glass spheres Mules)	52	9.6
Berens and Rosa*	(Rosa hollow plastic)	72	.06

* Cases include those of Berens and Rosa as well as numerous ophthalmologists throughout the United States who coöperated in the research work.

evisceration using plastic implants and a technique with special nylon sutures might be of interest.

HISTORY

The abscission or amputation of the anterior segment of the eye was first performed by Critchett in 1865. The base of the staphyloma was pierced with four or five silk sutures, emerging on the opposite side of the staphyloma which was then resected with two small triangles of sclera on each side of the horizontal diameter. The sutures were drawn taut, and tied.

Since cases of suppuration were reported after Critchett's procedure with danger of sympathetic ophthalmitis, Knapp in 1868 suggested that the sutures be inserted through the conjunctiva and episclera, several millimeters from the limbus. The immediate complication of this procedure was hemorrhage with loss of vitreous and frequently the choroid and the retina presented in the wound.

Mules[1] introduced a hollow glass or gold sphere, 11 to 13 mm. in diameter, into the scleral shell. The technique he advocated included undermining the conjunctiva to the equator of the eye; curettage of the papilla to prevent irritation to the nerve; and the removal of two small triangles of sclera at each end of the vertical diameter of the wound. He believed that the results were more satisfactory when the sclera was closed vertically and the conjunctiva horizontally. Minor modifications followed Mules' technique and the main difficulty to overcome was the expulsion of the implant.

Fox[7] thought expulsion of the implant was due to muscular traction on the sclera and preferred to sever the tendon of the recti and include Tenon's capsule in the conjunctival suture.

Gifford[8] favored simple evisceration. He contended that evisceration with excision of the cornea was performed too frequently and advocated incision of the sclera between the superior and lateral recti. The incision was approximately 20 mm. in length through which the contents of the globe were removed.

In 1936, Poulard[9] demonstrated the superiority of hollow cylindric glass implants. Since plastic implants were used with success after clinical and experimental enucleation,[10] plastic intrascleral implants were made in the same size as the glass cylinder.

Prior to using cylindric plastic implants, plastic spheres were used by one of us (C. B.) in five cases of evisceration. The cosmetic results were fair to excellent, with no extrusions reported over a period of 12 years. Approximately 12 eviscerations were performed with solid plastic cylindric implants over a period of eight years with no extrusions reported to date.

The motility in most instances was superior to that in which spheres were employed, with less sinking of the upper eyelid and lagging of the lower eyelid. The plastic cylinders 9.0 by 16 mm. were originally made solid, but have recently been made hollow to reduce their weight.

It has become apparent that extrusions of the implant are due to (1) the implantation of spheres which were too large, (2) intrascleral hemorrhages that had not completely ceased before insertion of the implant or developed later, and (3) scleral sutures that were not strong enough to withstand the distention in cases of scleral hemorrhage and edema followed by contraction of the sclera.

Research is being conducted on a new type

of intrascleral implant devised by Rosa, which is spherical on its posterior half (9.0-mm. radius) and with a flattened anterior surface. A flat-backed prosthesis is worn and better motion is claimed to be imparted by the flattened anterior surface of the stump than is obtained when the posterior surface of the prosthesis and anterior surface of the stump are rounded. Further results with these implants will be reported.

Indications for evisceration

a. Traumatic lesions of the eyeball, including intraocular foreign bodies and rupture of the globe

b. Perforation of large corneal ulcers

c. Mild atrophy of the globe following trauma

d. Chronic uveitis appearing in eyes that have been injured a long time previously or after intraocular surgery

e. Corneal and scleral staphylomas with glaucoma and disfigurement

f. Glaucoma secondary to uveitis or trauma in which surgery failed

g. Buphthalmos

h. Chronic endophthalmitis of low severity

i. Inactive absolute glaucoma

j. Unsightly eyes, damaged from various causes

Contraindications

a. In cases of tumor

b. When sympathetic ophthalmitis is present or is feared

c. Advanced phthisis bulbi with degeneration

Instruments

a. Eyelid speculum

b. Narrow cataract knife

c. Curved corneal scissors

d. Fixation forceps

e. Curette

f. Evisceration spoon

g. Strong straight scleral scissors

h. Plastic compressor[11]

i. Illuminated retractor[12]

j. Needle holder

k. Six double-armed (5-0) blue nylon sutuers (D & G #1648*)

l. Plain catgut (5-0) for the conjunctiva

Anesthesia

a. General anesthesia especially in painful eyes

b. Local anesthesia includes instillation, subconjunctival infiltration, and retrobulbar nerve block

Sterilization of plastic implant

a. Place plastic implant in aqueous zephiran 1:3,000 or oxycyanide of mercury 1:5,000 for 20 minutes. Alcohol or boiling water attacks plastic and should not be used.

Technique

First stage: Conjunctival incision

a. The conjunctival and subconjunctival tissues are picked up at the limbus with forceps and a complete circumcorneal incision is made one mm. from the limbus.

b. Using Stevens scissors, the conjunctiva is undermined as far back as the insertion of the rectus muscles.

Second stage: Scleral section and evisceration

a. A scleral section, which includes one half of the circumference of the eyeball, is made with a narrow cataract or curved glaucoma knife (fig. 1).

b. The edge of the sclera is grasped at one extremity of the incision and the cornea, with one mm. of sclera, is completely excised with curved scissors (fig. 2).

c. An evisceration spoon is inserted between the choroid and the sclera and by gentle lateral movements all adhesions are broken as far back as the optic nerve.

d. The uvea is detached from the optic nerve with the evisceration spoon, and the entire contents of the eyeball are evacuated. If, in traumatic cases, the surgeon finds that

* Davis & Geck, Brooklyn, New York.

scleral scars have become pigmented, it is advisable to excise these scars and suture the edges of the wound with double-armed 5-0 blue nylon sutures.

e. Additional curetting is made at the level of the optic disc to bring it to the same level as the surrounding sclera to prevent secondary irritation of the nerve by the implant.

f. With the aid of an illuminated retractor, the scleral shell is carefully examined; if any of the choroid is still adherent to the sclera, it is thoroughly curetted with the spoon.

g. To obtain hemostasis, adrenalin-soaked gauze is kept in the scleral cavity with compression from the plastic compressor.[11] To prevent late hemorrhage, thrombin in isotonic saline solution, 500 to 1,000 units per cc., may be applied to the internal surface of the eye.

THIRD STAGE: PLACING THE IMPLANT

a. A triangular excision of the sclera is made at the horizontal extremities of the scleral wound to transform the circle into an elongated opening to facilitate good clo-

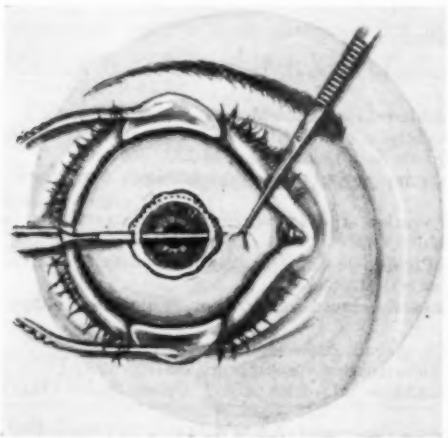

Fig. 1 (Berens and Rosa). After incising the conjunctiva as close to the cornea as possible and undermining it, a cataract knife is passed in the horizontal meridan one mm. posterior to the limbus and the incision is carried upward as shown by the dashed line.

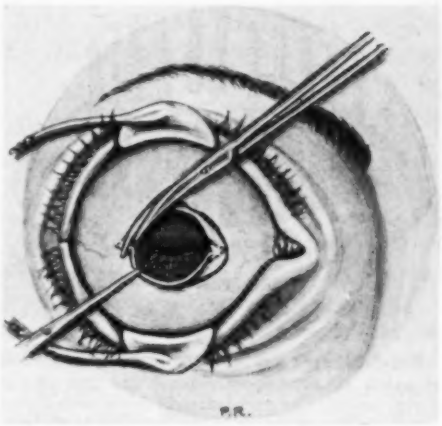

Fig. 2 (Berens and Rosa). The corneal excision is completed keeping in the sclera at least one mm. posterior to the limbus.

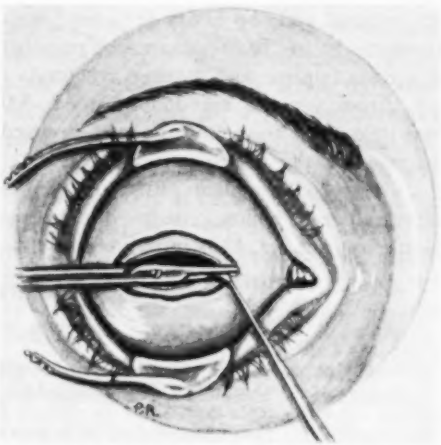

Fig. 3 (Berens and Rosa). Wedge-shaped pieces of sclera are excised with scissors at the ends of the horizontal diameter of the scleral wound.

a b c

Fig. 4 (Berens and Rosa). Hollow plastic intrascleral implants. (a) Hollow plastic sphere (Berens). (b) Hollow plastic cylindric implant (Berens). (c) Hollow plastic implant (Rosa).

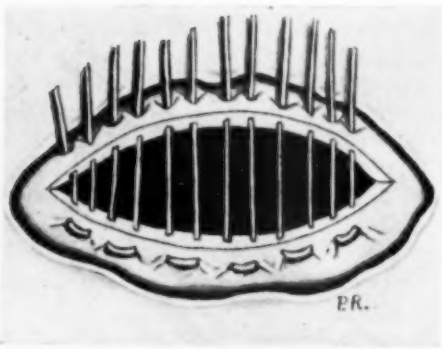

Fig. 5 (Berens and Rosa). Six mattress sutures of nylon are introduced through the lips of the scleral wound. The conjunctiva is closed with a running plain catgut suture which is locked in the center and at the extremities of the wound.

sure of the scleral wound (fig. 3). In the presence of a panophthalmitis, the sclera is not sutured and the cavity is packed with antiseptic gauze. In the absence of purulent infection, implant and sutures are used.

b. From four to six double-armed, 5-0 blue-nylon, mattress sutures are inserted through the edge of the scleral lips.

c. The plastic implant (Rosa plastic implant or Berens hollow cylindric or spherical plastic implant, fig. 4) is introduced into the scleral cavity and the sutures tied (fig. 5).

Discussion and Summary

Reviewing the literature on reported cases of sympathetic ophthalmitis following evisceration particularly a half century or more ago, revealed that the concept of this disease was entirely different from that held at present. There is sufficient evidence that evisceration is no more likely to be followed by sympathetic ophthalmitis than enucleation. Either operation may be futile if performed too late.

The amplitude of motion of the prosthesis following evisceration with Berens's hollow plastic cylindric or spherical implants, and especially with special Rosa intrascleral implants, is greater than that obtained with buried implants after enucleation. The technique is simple and surgically less traumatic than enucleation, causing less atrophy of the orbital tissues and consequently less sinking of the upper eyelid.

Evisceration is preferable to enucleation as a means of obviating the danger of meningitis from cutting the optic nerve in the presense of a purulent infection.

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References

1. Mules, P. H.: Evisceration of the globe, with artificial vitreous. Tr. Ophth. Soc. U. Kingdom, **5**:200, 1884-1885.
2. Van Lint, A.: L'exentero-enucleation du globe oculaire. Bruxelles-Medical, **19**:1206, 1938-39.
3. Report of the Committee of Ophthalmological Society, appointed in March, 1896, to consider the relative value of simple excision of the eyeball. Tr. Ophth. Soc. U. Kingdom, **18**:233, 1897-98.
4. Weekers, L.: Les enseignements de la guerre au sujet de l'ophthalmie sympathique. Ann. d'ocul., **154**:196, 1917.
5. Verrey, L.: Cinq cas d'operation de Mules. Evisceration et insertion d'un globe d'argent dans la sclerotique. Bull. et. Mem. de la Soc. franc. d'ophtal., **16**:164, 1898.
6. Burch, F. E.: Discussion (on previous paper by Pfeiffer, R. L.: The effect of enucleation on the orbit). Tr. Am. Acad. Ophth., **49**:237, 1944.
7. Fox, L. W.: Present status of Mules' operation and delayed implantation of gold balls. Ophthalmoscope, **7**:6, 1909.
8. Gifford, H.: On strictly simple evisceration of the eyeball. Arch. Ophth., **29**:422, 1900.
9. Poulard, A.: Insertion sclerale de spheres et ovoides solides. Ann. d'ocul., **173**:120, 1936.
10. Berens, C.: Synthetic plastic material for implantation into orbit. Am. J. Ophth. **24**:550 (May) 1941.
11. ———: A plastic compressor for enucleation and evisceration. J.A.M.A., **149**:1316 (Aug.) 1952.
12. ———: An illuminated retractor for eye operations. Am. J. Ophth., **27**:281 (Mar.) 1944.

CLINICAL DATA CONCERNING THE CENTRAL ORIGIN OF GLAUCOMATOUS ATTACKS*

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One characteristic symptom of a glaucomatous attack is a sudden dilatation of the pupil. Since the center which regulates dilation of the pupil is located within the diencephalon, one may assume that pupillary dilatation during a glaucoma attack is not an isolated phenomenon but is related to other dysfunctions of vegetative nature.

The present investigation frequently revealed a sudden rise in blood pressure (table 1) during an attack of glaucoma, frequently on the side of attack (Horniker). This peculiarity is especially conspicuous if the patient is examined a short time after the attack. The seven cases summarized in Table 1 came under observation one to six hours after the attack; four of them showed sudden rise in blood pressure which disappeared some days later.

Concomitant pupillary dilatation and increase in blood pressure point to an irritation of the central sympathetic nervous system. The pupil would also be dilated if the parasympathetic system were paralyzed but in such a case there would be paralysis of accommodation as well.

This difference is illustrated by the case of a woman, aged 48 years, who was examined one hour after an acute attack of glaucoma in the right eye. Vision was 5/20. Accommodation of the involved eye was the same as that of the unaffected left eye. It is worthy of mention that, during the prodromal stage, the patient complained of veiled vision and headache, especially if she had been reading or sewing for some time.

The effect of darkness on the retinal-hypophyseal reflex well illustrates the ocular-diencephalic relationship, as the following case shows:

A woman, aged 50 years, suffered from hypophyseal obesity and hyperostosis frontalis interna (Morgagni's disease). She complained of prodromal glaucomatous signs. After one hour in a dark room, the intraocular pressure of the right eye increased from 20 mm. Hg (Schiøtz) to 80 mm. Hg. The intraocular pressure in the left eye changed scarcely at all. The diameter of the pupil of the right eye showed almost no alteration and the angle, viewed gonioscopically, remained unchanged in spite of the stress. This would indicate that the greatly increased pressure following subjection to darkness was due to a central reflex and was not a sequence of local alteration.

To prove this, the retrobulbar ciliary ganglion was blocked by an injection of

* From the Ophthalmological Department of the Stiller-Bertalan Hospital.

TABLE 1

BLOOD-PRESSURE FINDINGS IN CASES OF GLAUCOMA DIAGNOSED SHORTLY AFTER AN ACUTE ATTACK

Name	Sex	Age (years)	Diagnosis	Eye	Blood Pressure (mm. Hg)	
					Right	Left
K. S.	F	83	Acute glaucoma	O.D.	200	210
F. W.	F	62	Acute glaucoma	O.D.	240	210
F. S.	M	53	Acute glaucoma	O.S.	140	160
M. I.	F	64	Acute glaucoma	O.S.	220	215
W. I.	F	68	Acute glaucoma	O.S.	150	180
I. H.	F	50	Acute glaucoma	O.D.	155	140
G. S.	M	66	Acute glaucoma	O.S.	150	150

TABLE 2

THE EFFECT OF STELLATE GANGLION BLOCK

Name	Sex	Age (years)	Side of Block	Ocular Tension Change (mm. Hg)		Blood Pressure Change (mm. Hg)
				Right	Left	
G. K.	F	60	Right	19–20	18–18	165/90 135/85
L. E.	M	31	Left	21–21	23–26	
L. R.	M	43	Both	19–23	19–23	170/100 122/90
N. S.	M	60	Right	23–30	23–23	145/80 125/75
I. S.	M	57	Both	21–23	21–23	160/95 130/80
J. N.	M	64	Right	22–24	22–22	

novocaine, which resulted in ptosis, mydriasis of medium degree, and paralysis of accommodation. In spite of a one-hour stay in a dark room, the patient's intraocular pressure remained unaltered, at 20 mm. Hg. After retrobulbar block which excluded the peripheral ganglion, the response of the retinal-hypophyseal reflex to darkness did not extend through the efferent pathways to affect the intraocular pressure.

The results of studies of Horner's phenomenon point to a neurovascular regulation of the ocular tension. In several cases, after stellate-ganglion block with novocaine for other diseases, the ocular tension was taken. After ganglion blockage had been demonstrated by the appearance of Horner's triad, the ocular tension and blood pressure were determined (table 2).

In every case, there was a slight increase in intraocular pressure on the side blocked. Although the readings in all but one case (Case 4) remained within the limits of error, the results were always perfectly clear. Blood pressure decreased in each case. After Horner's phenomenon disappeared, the intraocular pressure returned to its original value.

If ocular tension is taken years later in cases in which Horner's triad was a sequence of trauma or surgical intervention, it will be found to be considerably lower on the involved side (Filbry). Although it is easy to understand the increase in ocular tension directly associated with Horner's triad as a consequence of sympathetic paralysis, it is more difficult to explain the lowering after considerable lapse of time. It may be that the altered function of the denervated vascular wall is of significance.

SUMMARY

Acute attacks of glaucoma are frequently associated with sudden increase in blood pressure. Maximum dilatation of the pupil with unaltered accommodation is a sign of central sympathetic irritation.

Response to the dark-room test is a retinal-hypophyseal neurovascular-intraocular reflex; in spite of sudden increase in ocular tension, the angle, viewed gonioscopically, is unaltered.

After ciliary ganglion block, response to the darkroom test becomes negative in cases in which it formerly was positive. Blockage of the stellate ganglion (appearance of Horner's triad) directly increases the ocular tension and decreases the blood pressure.

In cases in which Horner's phenomenon has been present for years, intraocular pressure is decreased on the affected side.

V., Szemelynok-u. 9-11. ½3.

ANALYSIS OF RESULTS WITH THE MASSACHUSETTS VISION TEST*

WITH RECOMMENDATIONS FOR IMPROVING ITS ACCURACY

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A recent study by Yasuna and Green[1] pointed out certain deficiencies in the Massachusetts vision test as applied to school children. Their material consisted of written reports from several different specialists and oral reports from parents, making the total 161. The results were evaluated on the basis of whether or not each child was given a prescription for glasses. Of the entire group, 65 percent were given glasses, 35 percent were not.

Yasuna and Green advised that the younger children (Grades 1 to 3) be given the 20/30 line as passing on Part I and lenses of +2.25 diopters for Part II. The older children (Grades 4 to 7) should read 20/20 for Part I and use lenses of +1.75 diopters for Part II. They felt that Part III of the test (Maddox rod) could be eliminated entirely.

In the present study, an attempt was made to eliminate the necessity of reliance upon written reports from scattered examiners. A second screening† of the children, who twice failed the Massachusetts vision test as given by a school nurse, was carried out in the DeKalb County Health Department Eye Clinic by two assistants and me.

The procedure was as follows:

1. Recheck of visual acuity by the Snellen type illiterate "E" chart and a number chart.

2. Retinoscopy with +1.5D. sph. and +3.0D. sph. for an estimation of type and amount of refractive error.

3. Cover-uncover test for distance and near. Maddox-rod and prism tests were done on all children who had failed Part III of the Massachusetts test.

4. Ophthalmoscopic examination in cases of suspected intraocular pathologic conditions.

MATERIAL

The Massachusetts vision test was administered to 2,226 school children by the DeKalb County Health Department staff of nurses. There were 410 children who failed the test on two occasions (table 1). The failures were 18.4 percent of the total, a figure approximating that found in other studies.[1, 2] Part I of the test accounted for 37.5 percent of the total failures, Part II for 52.5 percent, and Part III for 10 percent.

TABLE 1

RESULTS OF VISUAL SCREENING TEST

No. Tested	2,226
No. 2nd Failures	410 (18.4%)
Part I—37.5%	
Part II—52.5%	
Part III—10.0%	

RESULTS

Of the group of 304 children who failed the school test and were brought to the clinic for further studies, 163 or 53.6 percent were classified as not needing further study, and 141 or 46.4 percent were referred for definite eye care. The accuracy of this final classification was high as evidenced by the fact that 92.5 percent of the referred children who were examined were found to need glasses or some form of treatment. The results for each part of the test are seen in Table 2.

ANALYSIS

In Part I, 20/30 was considered as passing for children in the first three grades of

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† Dr. Albert Fisher, Jr., and Dr. Norman F. Stambaugh, Jr., assisted in the final screening process.

TABLE 2

RESULTS OF FINAL SCREENING BY OPHTHALMOLOGIST OF CHILDREN FAILING SCHOOL TEST

Grade	Part I			Part II			Part III		
	No.	Passed	Failed	No.	Passed	Failed	No.	Passed	Failed
1	19	8	11	21	15	6	3	1	2
2	22	6	16	34	26	8	5	2	3
3	12	2	10	30	24	6	4	2	2
4	17	3	14	31	25	6	6	2	4
5	13	1	12	15	12	3	3	1	2
6	17	5	12	14	12	2	5	2	3
7	14	1	13	15	11	4	4	2	2
	114	26 (22.8%)	88 (77.2%)	160	125 (78.1%)	35 (21.9%)	30	12 (40.0%)	18 (60.0%)

school. Most of the children who failed this part of the test at school and later passed it at the clinic were in the younger group. They had been confused or frightened by the test but, with reassurance and coaxing, they later read the symbols correctly. Even with this source of error, Part I of the Massachusetts vision test was found to be 77.2 percent accurate. With more help and explanation to the younger children, the results can be improved further.

A large number of children who failed Part II of the test were later classified as passing. In fact 78.1 percent of the failures were passed by the clinic test. There was no great difference observed between the younger and older children in this test.

There were two reasons for the discrepancy between the two screening tests for hyperopia. First, the schools considered the ability of any child to read 20/30 through the +1.50 diopter lenses as failure, whereas we used 20/20 for the older students. Secondly, we felt that +1.50 diopters were not sufficiently strong because, even though a child could read the symbols through such a lens, retinoscopy did not reveal a refractive error of that magnitude. On questioning many of the children, we found that they were seeing the symbols as a blur but could still detect their correct position.

Part II of the test can be improved by using +2.25 diopter lenses for the younger children and +1.75 diopter lenses for the older children.

Ten percent of the total failures belonged to Part III. We found that 40 percent of this group had a normal muscle balance and that 60 percent had some motor anomaly. There was no difference observed between the younger and the older age groups. It is felt that Part III of the test is acceptable in its present form. Because the number of failures is rather low (10 percent of the total failures or 1.8 percent of the total test group) and because it is more time consuming than Parts I and II together, it could be omitted when large numbers of children need to be tested and there is a shortage of trained personnel to administer the test.

RECOMMENDATIONS

On the basis of the findings herein presented, the following changes in the Massachusetts vision test are recommended:

Part I —Grades 1-3 20/30
Grades 4-7 20/20
Part II —Grades 1-3 +2.25D. sph.
Grades 4-7 +1.75D. sph.
Part III—No change.

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REFERENCES

1. Yasuna, E. R., and Green, L. S.: Am. J. Ophth., **35**:235-240, 1952.
2. Sloane, A. E.: Arch. Ophth., **24**:924-939, 1940.

SUBMUSCULAR SCLERECTO-CYCLODIALYSIS*

A NEW TECHNIQUE FOR AN ANTIGLAUCOMA OPERATION

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Of all the operations proposed for ocular hypertension, cyclodialysis is perhaps the most rational. It only slightly alters the structure of the eye; it is not dangerous to the eye; and it is easy to execute and has wide application since it can be used for the different varieties of glaucoma. The main argument against it is the inconstancy of its results, especially with the passing of time, and its minor hypotensive action if compared with other methods.

Several modifications have been proposed for Heine's original method (1905) with a view to improving the operative act, to prevent immediate accidents, and to obtain good definite results. Among the modifications are those that aim at better fixation of the eyeball during the surgical procedure, those that are mainly concerned with the manner of introducing the spatula, with the quadrant on which to operate, or with prevention of immediate hemorrhages into the anterior chamber. Some of these procedures have undoubtedly proved their efficacy and deserve general approval.

Moved by the wish to contribute to the perfection of the technique in this procedure, I have devised a new type of cyclodialysis, the *sclerecto-cyclodialysis*. A discussion of its manner of action and results will enable us to draw some physiopathogenic considerations further on.

Heine[1] practiced cyclodialysis on the inferoexternal quadrant of the eyeball, using an arched spatula graduated in millimeters, having previously cut the sclera with a keratome following an oblique line to favor its introduction. Once the end of the instrument reached the anterior chamber, he rotated it 90 degrees to each side in order to obtain the desired dialysis.

Elschnig,[2] a great advocate of cyclodialysis, favored the practice of the conjunctival incision at a greater distance from the limbus, thus setting the scleral area free so that this area could be used in turn as a resting point for the forceps used for the scleral fixation. He introduced the spatula which is now the most widely used, although the best spatula in my opinion is Stallard's.[3]

Elschnig[2] recommended a greater sweep of the spatula, thus obtaining a wide separation of the ciliary body. To prevent an immediate hemorrhage, he ordered his assistant to compress the equatorial region of the eyeball on the opposite end of the meridian in which intervention had been made. If the blood reached the anterior chamber in spite of this measure, he introduced a Daviel spoon through the opening for its evacuation (a dangerous procedure), in which case he performed paracentesis several days later. It should be made clear that these measures have proved impracticable in many cases.

The difficulty presented by the introduction of the spatula into the anterior chamber, due to the presence of the scleral torus, was overcome by Blaskovicz[4] with his inverse cyclodialysis which consisted of introducing the spatula not directly toward the anterior chamber but paralleling the limbus and then making it turn 90 degrees to reach the anterior chamber; the spatula is then removed and reintroduced and, starting from the other edge of the incision, the step is repeated.

The Hungarian professor maintained that none of the quadrants of the globe should be used to reach the sclera but that it should be approached directly in front of the superior rectus. The incision should be made in

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the direction of the meridian that passes through the center of this muscle. The fixation of the eyeball obtained by using the tendon as a support for the forceps renders the maneuver with the spatula much easier.

A similar procedure is advised by Gát[5] who operates immediately in front of the external rectus which he previously has isolated and securely fixed by means of a myostat.

With the name *transcorneal cyclodialysis*, Campos[6] has tried to simplify cyclodialysis by means of a method which could more correctly be termed "inverse" than that of Blaskovicz.

As its name indicates, in Campos' operation the incision is made on the cornea itself, three mm. inside the corneal limbus; the spatula is introduced through that incision in a centrifugal direction with the purpose of "breaking" the chamber angle and sliding it against the sclera, the ciliary body being separated from it. According to its author, the advantages of this procedure are its rapidity and the absence of subconjunctival hemorrhage.

Desiring to obtain immobility of the eyeball and to prevent scleral hemorrhages and hyphemas, Danielson, Long, and Sherwood[7] introduced several modifications, consisting of previously placing two parallel sutures on the sclera very close to the incision, of cauterization of the sclera and injection of air into the anterior chamber as devised by Randolph.[8] In my opinion this last measure constitutes one of the great progresses in the operation.

The tendency, erroneous in my opinion, to try to find a greater or more lasting ocular hypotonia has prompted several surgeons to modify Heine's original operation or to combine it with other antiglaucomatous interventions. Thus we have suprachoroidal iridotasis (Mauksch[9]); cyclodialysis with iridectomy (Wooton[10], Wheeler[11]); cyclodialysis with iridencleisis (Del Barrio[12]); limbal sclerectomy cyclodialysis (Cornet[13]); iridosclerecto-cyclodialysis (Queiroga[14]); the introduction of magnesium into the opening produced by the spatula (Troncoso[15]), and so forth.

None of these variations coincides with the method herein presented nor with its theoretic fundamental principles. As so often happens, almost always the idea one thinks original someone has already put into practice. This has happened to me. Once I performed the operation and verified its good results, I found out that Römer[16] and Sallmann[17] had already thought of similar operations.

Römer's operation consists in a retrociliary transmuscular trephination, planned for absolute glaucomas or as means of subretinal drainage. Sallmann's operation is a cyclodialysis with trephination, performed in the ciliary region.

A still greater similarity is found in one of the methods proposed by Cornet[13] and that of Mügge.[18] Cornet's retrociliary sclerectomy-cyclodialysis is performed by practicing a scleral incision with the sclerotome, at five or six mm. from the limbus, afterward partially resecting its anterior border with a hollow punch. In Mügge's method, scleral trephination is practiced 1.5 to 2.5 mm., seven or eight mm. behind the limbocorneal region on the inferior scleral quadrant. Cornet prefers to work on the superior quadrant.

Operative technique

Anesthesia. Myosis is produced with eserine and an injection of 1.5 cc. of two-percent novocaine is used for retrobulbar anesthesia. Subconjunctival anesthesia is obtained by injecting 1.0 cc. of two-percent novocaine near the tendon of the external rectus.

Incision of the conjunctiva is made parallel to the limbus at the tendon of the external rectus and the tendon is exposed on its superior border and lifted with a strabismus hook. The border of the tendon, close to its scleral insertion, is lifted with a double hook (fig. 1-a) that replaces the strabismus hook.

The underlying sclera is exposed, and, if desired, may be cauterized with a red-hot ap-

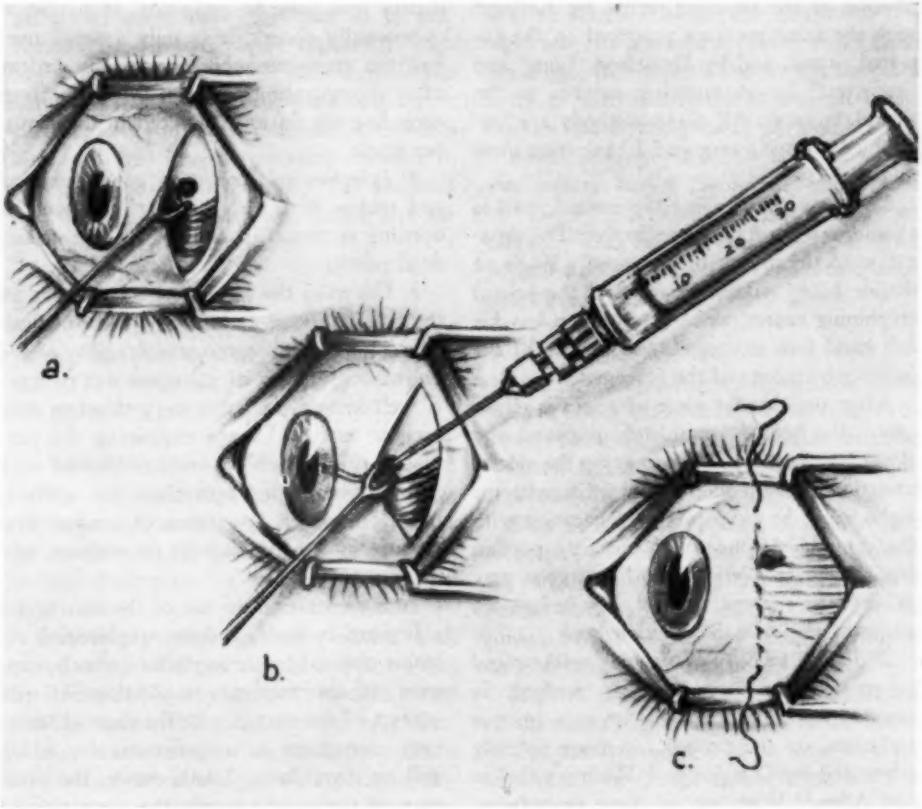

Fig. 1 (Malbrán). Technique of submuscular sclerecto-cyclodialysis. (a) The border of the tendon is lifted with a double hook close to its scleral insertion. (b) The cannula is introduced and the cyclodialysis is performed with this instrument. (c) The conjunctiva is closed with a continuous suture.

plicator to prevent hemorrhage, but this measure is, in my opinion, unnecessary.

With the eyeball held firm with forceps, the surgeon is able to grasp the tendon at its scleral insertion and a 2.5-mm. or 3.0-mm. trephine is used to perforate the sclera. Although the underlying choroid may or may not herniate, it is well to avoid herniation by relaxing tension and pressure on the tendon.

The cannula of Randolph, modified by Castroviejo (Meyrowitz), is introduced on a Luer-lock syringe (fig. 1-b) and the cyclodialysis itself is then performed with the same instrument, preferably in the manner of Blaskovicz (inverse cyclodialysis). I do not use Elschnig's nor any other type spatula.

Air injection. After the dialysis is performed and while the end of the cannula is in the anterior chamber, a bubble of air is injected through it to avoid the possibility of hyphema. The cannula is rapidly withdrawn and the conjunctiva is closed with a continuous suture (fig. 1-c). The scleral opening is covered with the muscle. A binocular bandage and pilocarpine are applied. The patient is kept in a semi-upright position.

Discussion of the procedure

The advantages and disadvantages of the procedure may be described by comparing this operation with the different types of cyclodialysis previously proposed.

1. *The fixation of the eyeball* during the operation is surgical. Heine's original method was improved by Blaskovicz[4] with

fixation of the superior rectus, by Kettesy[5] with the same measure practiced on the external rectus, and by Danielson, Long, and Sherwood[7] by juxtaposing sutures to the scleral incision. All these methods are undoubtedly satisfactory and I have used them all successfully.

My means of keeping the eyeball fixed is simple, safe, and no less effective. The separation of the muscle by means of a single or double hook makes initiation of the scleral trephining easier, since the surgeon has his left hand free to complete separation of the muscles by means of the forceps.

After the circular piece of sclera is eliminated, the hook is completely removed and the surgeon uses forceps to grasp the scleral insertion of the tendon firmly while, with his right hand, he performs the operation with the cannula. If the scleral circular portion has been incompletely cut, the surgeon may finish with forceps and scissors before his assistant removes the traction hook.

2. *The possibility of meeting with scleral torus,* as occurs in Heine's method, is avoided by following Blaskovicz's inverse technique or the procedures more recently advocated by O'Brien and Weih[19] and Lee and Allen.[20] With any of these procedures, it is possible to obtain a wide ciliary dialysis that reaches, if necessary, as far as half of the circumference. With my technique, wider posterior dialysis is possible, since in my method, the separation of the ciliary body starts from further behind, producing more complete anteroposterior coverage.

3. *Hemorrhage* in the anterior chamber is easily avoided with the injection of air, a fact which has already been verified by Sugar,[21] Shaffer,[22] Barkan,[23] and others. Nevertheless, my way of directing this step of the surgical operation represents a technical improvement.

Hyphema is less likely to take place if the air is rapidly injected into the anterior chamber after the dialysis. If the same instrument is used for dialysis and injection of air, the time of intervention takes scarcely more than a minute, with the immediate benefit that may be imagined. If hyphema occasionally occurs, it is only a small one, and the semisitting position of the patient after the operation prevents the blood from occluding the fissure produced in the chamber angle.

4. *Injection of air* avoids goniosynechias and makes it much easier to maintain the opening or tunnel just completed, with beneficial results.

5. *Covering the trephination* by means of the muscle (fig. 1-c) is simple and practical. The thinness, adherence, and fragility of the bulbar conjunctiva of glaucomatous patients is well known, and this very thinness may become extreme, hence explaining the precarious and rather late results obtained with Elliot's trephining operation. In addition there are not a few cases of conjunctival rupture and infection with or without rupture.

In my operation the use of the muscle is a safeguard in itself, and the trephination remains covered by three planes (muscle, capsule, and conjunctiva). In addition, intimate contact of the muscle with the choroid seems very convenient to me—reasons for which will be given later. Furthermore, the presence of the muscle avoids the formation of scar produced by the Lagrange and Elliot methods, a scar which, in some cases, develops to such an extent that it causes the sensation of a foreign body in the upper eyelid and produces lacrimation.

6. *The operation is extremely simple and efficacious.* It requires few instruments and can be adapted to almost all the varieties of ocular hypertension. It can be repeated at least twice on each muscle.

7. *Ciliary body.* There is no danger of injuring the ciliary body in making the scleral incision, as may occur when other methods of cyclodialysis are used. Although this accident seldom happens to experienced surgeons, in my procedure it is absolutely impossible, since the scleral trephination is made at the same level as the pars ciliaris retinae.

What has happened to me instead is that,

in many cases, once the choroid is perforated, the vitreous humor appears through the trephine opening. This little accident has happened without any later complications; it usually occurs in cases of aphakic eyes when air is injected into the anterior chamber in too great a quantity, or by the assistant exerting too much pressure during the execution of the trephination.

Results

The success of the operation must be judged by (1) its immediate and late results; (2) its manner of action; (3) cases for which it is indicated.

1. Immediate and late results.

The hypotonia that immediately follows the intervention is replaced in some cases by an elevation of the ocular tension which usually disappears on the second or third day.

This event, which frequently occurs in all types of cyclodialysis, has been pointed out by Heine,[1] Blaskovicz and Kreiker,[4] and Gát[5] in their publications on the operation. Rather late normalization of ocular tension is, perhaps, the neuralgic effect of the cyclodialysis, since the tendency to lose the beneficial results as time goes by is a well-known fact. I shall not deal with this aspect of the problem here, since I still lack the experience based on long-time observations.

Immediate reduction in visual acuity may be due to hyphema, the rupture of Descemet's membrane, or postsurgical myopia.

I have already described how the injection of air into the anterior chamber prevents the hyphema, the true "bête-noire" of the time of Heine and Elschnig, who did not resort to the use of air injection.

The ruptures or lacerations of Descemet's membrane are produced by the traumatic action of the spatula; however, they can be easily avoided with Blaskovicz's inverse technique. If Castroviejo's spatula-cannula is used, that possibility is less probable. The gold color of the instrument makes it easily detected through the transparent tissues under the sclera, enabling one to control it by sight until its free end apppears in the chamber angle; that control also avoids the possibility of introducing the instrument behind the iris and injuring the crystalline lens.

Immediate myopia following the operation was pointed out by Heine[1] and was verified by Salus,[24] Stein,[25] Bunge,[26] Gát,[5] and Sugar.[21] This myopia generally persists for several weeks or months. It is due to diminution of tension on the zonular fibers.

2. Manner of action.

Heine's[1] original opinion was that cyclodialysis opens a new canal of excretion to the aqueous humor by opening a communication between the anterior chamber with the suprachoroidal space.

This theory was opposed by Salus[24] who supported the view that the diminution of the ocular tension was the consequence of partial atrophy of the ciliary body. He was joined in this opinion by Kraus, Schnabel, and Meller, among others (in Heine).

Gradle[27] maintained that the happy result of the operation was due only to the release of the chamber angle that permits the aqueous humor to escape through the canal of Schlemm.

Although Kraus' investigations on cats and rabbits proved that the intervention was followed by the late appearance of a solid scar on the operative site, his argument is of little value, since other scientists, namely Elschnig,[2] Loddoni,[28] and Stoutenborough,[29] have encountered no atrophy of the ciliary body and that is the fundamental anatomic principle on which Salus, Kraus, and others base their opinion.

Wernicke,[30] who had joined these gentlemen in the interpretation of the manner of action of the cyclodialysis, gave his opinion in advance, saying that the problem could not be solved through studies based on animals.

Opinions are also divided as regards experiences with human eyes, the anatomic study of which was possible after surgical failure. While some have denied the formation of a fistula in the chamber, others have

failed to prove the existence of a wide ciliary atrophy.

More recently, gonioscopic studies have permitted definite acceptance as correct of Heine's[1] hypothesis on the formation of a fistula in the chamber. Vannas[31] first, Barkan, Boyle, and Maisler,[32] Clarke,[33] and Sugar[21] later, have established that the existence of the fistula is a "sine qua non" condition to obtaining lasting hypotonia. It must not be forgotten, however, that there are some exceptions to this rule—the few cases of normal ocular tension after closing of the fistula. The existence of the fistula in the chamber explains the hypotonia by communication between the anterior chamber and the suprachoroidal space; the posterior uveal tract thus creates a true posterior drainage.

Gonioscopic observation by means of Goldmann's lens has convinced me that a fistula in the chamber is the object of all correct cyclodialyses. Furthermore, when cyclodialysis is finished with the injection of air, although the air bubble is not reabsorbed, the root of the iris and its parts near the fistula are depressed, resulting in a greater depth of the anterior chamber in that region. Gonioscopy is not needed to make this observation.

This verification indicates that the classical cyclodialysis and some of its modifications actually become procedures of internal fistulization as opposed to the external fistulization created by the procedures devised by Lagrange, Elliot, and others, which are all operations that may be classified as "juxtalimbar." Only Sallmann's, Cornet's, and especially Mügge's methods are true examples of retrociliary external fistulization.

To these last must now be added the method herein advocated, if we accept maintenance of the trephine opening as the beneficial factor. In this connection, it must be remembered that the fundamental objection to posterior sclerectomy in operations for glaucoma is the facility with which cicatrization takes place, and there is no lasting hypotonic action, such as occurs in Lindner's[34] operation. I believe that my method will give better results because:

In the first place, though hypotonia may be produced with a trephination less than 1.5 mm. in diameter, I think it convenient to make an opening of 2.5 or 3 mm.; this implies no risk at all and the opening remains protected by the muscle. Besides, *it is probable* (anatomic studies are necessary to prove it) that the formation of a vascular plexus between the muscle and the choroid favors the action of drainage or external fistulization, an action which, being perhaps favored by the continuous movement of the muscular belly, may also prevent definitive closing of the scleral opening. These improvements do not deny the beneficial concomitant action of an internal fistulization as it occurs in other cyclodialyses.

The originality of my idea can no longer be presumed since I have discovered that Mügge had already conceived an almost identical operation with excellent results; the only difference between our methods is my use of the submuscular position and the wider diameter of the trephination. Time will say if this variation is or is not more effective than Mügge's procedure. For the present, I think it safer.

3. Cases for which the operation is indicated

Much has been written about the different types of glaucoma which may be improved through cyclodialysis.

Heine[1] considered this operation especially indicated in simple chronic glaucoma and even subacute glaucomas, but he believed that it became less effective as the ocular hypertonia became more acute; he ascribed to it a great therapeutic value for buphthalmos, some secondary glaucomas, glaucoma in the aphakic eye, and the glaucomas resulting from the luxation of the crystalline lens.

Barkan,[23] in his definite classification of the glaucoma of wide angle and that of shallow angle, maintains that the operation

combined with the injection of air is especially indicated for wide-angle glaucoma, and that it is the operation of choice in some cases of shallow-angle glaucoma with its peripheral adhesions and strong congestion because, under such circumstances, trephination, iridencleisis, or iridectomy are known to be of little value and associated with considerable risk. He adds that it may prove useful for some secondary glaucomas especially when other antiglaucomatous operations have failed.

Sugar[21] supports the view that the two "exclusive" cases for which the cyclodialysis is indicated are glaucoma in aphakic eyes and secondary glaucoma produced by the posterior luxation of the lens. It may prove successful for shallow-angle glaucomas in which previous iridectomy has failed. It is of little effect in cases of simple glaucoma, although it has some advantages in so far as the technical aspect is concerned.

For my part, I would make it clear that submuscular sclerecto-cyclodialysis has given results that can indeed be called extraordinary in aphakic eyes and in some secondary glaucomas in which the depth of the anterior chamber was greatly reduced. I have not had an opportunity to apply it in secondary glaucoma produced by the posterior luxation of the lens.

In simple glaucoma, the immediate results are very encouraging, superior in my opinion, to other surgical procedures. In buphthalmos I have used it in only one difficult case with good results. I plan to practice it systematically, although I must point out that Elliot's trephining operation has always given me excellent results in this type of glaucoma.

In subacute glaucoma, cyclodialysis according to the method herein described is excellent. I have not used it in hypertensive uveitis. In such cases I share Chandler's[35] opinion on the efficacy of Reese's puncture. At other times, iridencleisis seems to me the method to be chosen.

I agree in part with the opinion of those who support the belief that cyclodialysis is only useful when the chamber angle is wide; such was my criterion until I resorted to the injection of air. Indeed, the wide angle is a helpful detail; thus, Vogt's capsular glaucoma, almost always of wide angle, is improved by the operation.

Finally, let me say that acute glaucoma is generally well controlled by iridectomy, as Graefe taught; this has been my use in such cases, although posterior sclerotomy has of late proved of great efficacy. This subject shall be the theme of another publication.

Comments

The operation has been performed on 27 patients (36 eyes). Table 1 summarizes the results. In 19 cases of simple chronic glaucoma, there were two cases of aphakia (Cases 4 and 8). Among the eight cases of chronic glaucoma, Case 2 had been previously operated (iridectomy), and in Cases 5, 6, 13, 14, and 15, iridencleisis had been done without results. Only one case (Case 27) presented an enormous buphthalmos and the results in this case, as far as normalization of the tension was concerned, were excellent.

The visual acuity was improved or at least remained the same in every patient operated, except in Case 4 (turbidness of the vitreous, detachment of the retina), and Cases 5 and 7 in which, at present, cataract is present. There is no explanation of the partial loss of sight in Cases 11, 14, and 16.

In Case 19, although the patient's sight was excellent before the operation (7/10), his field of vision was extremely small and the fixation point was threatened. This sometimes happens following trephining operations or iridencleisis and would indicate that, in this condition, surgery may be contraindicated.

The most marked improvement of vision was obtained in eyes with corneal edema, with or without congestive conditions.

The tension was reduced in every case, although in some patients the intraocular pressure remained elevated above normal.

TABLE 1

SUMMARY OF THE CASES IN WHICH SUBMUSCULAR SCLERECTO-CYCLODIALYSIS WAS PERFORMED

Case	Age (years)	Diagnosis	Visual Acuity		Tension (mm. Hg) (Schiøtz)		Gonioscopy	Observations
			Before	After	Before	After		
1 R. G. M	49	Simple chronic glaucoma	R.E. 10/10 L.E. 8/10	10/10 10/10	25 30	20 20	O.U. Medium angle	14 days after the operation, the tension reached 20 mm. Hg and remained the same
2 H. G. M	50	Congestive chronic glaucoma	R.E. 2/10 L.E. 6/10	2/10 6/10	35 40	15 30	Very shallow angle. Dialysis well visible from 10 to 11 hr.	This patient had been operated on both eyes because of a hypertensive attack (total iridectomy in both eyes), postoperative hyphema in L.E., which was rapidly reabsorbed
3 M. N. F	44	Congestive chronic glaucoma	R.E. H.M. L.E. 5/10	H.M. 7/10	80 80	50 22	Very shallow angle; postsurgical dialysis from 2 to 2 hr.	The patient had been operated by iridencleisis, both eyes, and she had nonperforative cyclodiathermy applied to her R.E. with bad results. She was operated in two sessions
4 A. R. F	68	Simple chronic glaucoma Aphakia	R.E. 6/10 L.E. 3/10	6/10 Light	34 50	18 12	Wide angle; after the operation, it was observed that the dialysis was obstructed in the L.E.	Small hernia of the vitreous during the operation. Turbid vitreous after the operation (hyalitis, detachment of the retina)
5 B. V. M		Congestive chronic glaucoma	R.E. 4/10 L.E. 6/10	5/10 1/10	>90 >90	30 20	Shallow angle. After operation, it is observed that the dialysis in the L.E. is very small	The R.E. had been operated by iridencleisis without results. Postoperative diplopia lasting 3 weeks. The partial loss of sight in the L.E. was due to the formation of a corticonuclear-cataract
6 V. W. M	61	Congestive chronic glaucoma	R.E. 3/10 L.E. 10/10	1/10 10/10	30 22	25 25	Shallow angle. Dialysis with a small central synechia	The R.E. had been operated by iridencleisis 10 months before because of an acute glaucomatous crisis. Cyclodialysis with loss of vitreous humor and hyphema rapidly reabsorbed. The L.E. was not operated
7 M. A. F	66	Congestive chronic glaucoma	R.E. 1/10 L.E. 3/10	1/50 3/10	40 20	18 18	Shallow angle. Normal dialysis	Operation of both eyes in the same session, with no further complications. The partial loss of sight in the R.E. was due to the formation of a nuclear cataract
8 C. K. F	54	Simple chronic glaucoma	R.E. H.M. L.E. 1/50	H.M. 1/50	60 30	25 30	Shallow angle. Large synechia in the R.E.	R.E., aphakia; L.E., senile corticonuclear cataract; the cyclodialysis in the R.E. only
9 R. S. F	60	Simple chronic glaucoma	R.E. 1/10 L.E. 1/20	9/10 9/10	>90 >90	18 17	Shallow angle. R.E. ample dialysis; L.E. partially obstructed dialysis	The operation of the L.E. brought tension to 40 mm; patient operated again 5 months later with complete success. A torn piece of the Descemet's membrane was observed floating in the anterior chamber
10 A. M. M	49	Simple chronic glaucoma	R.E. 1/10 L.E. 1/10	1/10 1/10	60 27	22 26	Wide angle with gonosynechias; dialysis well visible	Large hyphema during operation which was totally reabsorbed. L.E. was not operated
11 E. A. F	67	Simple chronic glaucoma	R.E. H.M. L.E. 10/10	H.M. 5/10	30 34	30 18	Shallow angle. Small dialysis	Operated only on L.E.
12 A. V. F	44	Simple chronic glaucoma	R.E. 40/40 L.E. 1/10		10 10	21 20	Shallow Angle. Small dialysis	Persistent diplopia. Operated both eyes in two sessions
13 E. F. M	52	Congestive chronic glaucoma	R.E. 1/10 L.E. 1/10	8/10 8/10	55 75	18 18	Very shallow angel; very small double dialysis separated by a synechia	L.E. had an acute attack, hence iridencleisis one month before; the tension was reduced to 35 mm. Hg; since it did not continue to decrease, cyclodialysis was done

TABLE 1—(*continued*)

Case	Age (years)	Diagnosis	Visual Acuity		Tension (mm. Hg) (Schiøtz)		Gonioscopy	Observations
			Before	After	Before	After		
14 N. L. M	50	Simple chronic glaucoma	R.E. 3/10 L.E. 3/10	6/10 2/10	40 48	18 20	Shallow angle in both eyes. Dialysis of the R.E. was very long	L.E., iridencleisis 4 months before operation with excellent results. Cyclodialysis on L.E. only.
15 C. V. F	30	Congestive chronic glaucoma	R.E. 1/10 L.E. 6/10	7/10 10/10	>90 40	12 16	Shallow angle	Patient operated by iridencleisis, O.U., because of acute crisis of hypertension without improvement in the R.E. Cyclodialysis on R.E. only
16 J. P. F	54	Simple chronic glaucoma	R.E. 10/10 L.E. 0	7/10 0	34 65	20 30	Slightly shallow angle, O.U.	Only R.E. operated
17 M. N. M	60	R.E., simple chronic glaucoma L.E. atrophia bulbi	R.E. 7/10 —	9/10 —	40 —	20 —	Shallow angle	—
18 J. P.	59	Simple chronic glaucoma	R.E. 10/10 L.E. 7/10	10/10 7/10	30 34	20 20	Medium angle	Only L.E. operated in R.E., tension normalized with myotic drugs
19 J. G. M	48	Simple chronic glaucoma	R.E. 7/10 L.E. 10/10	1/10 10/10	55 20	15 20	Wide angle free	Only R.E. operated; very long dialysis from 8 to 11 hours. In spite of good field of vision before operation, the fixation point seemed threatened (surgical contraindication?)
20 A. F. A. M	75	Simple chronic glaucoma	R.E. Light L.E. 4/10	Light 4/10	55 55	55 27	Medium angle with gonio synechias	Only L.E. operated; postoperative seclusion of the pupil; very short dialysis
21 D. U. M	67	Simple chronic glaucoma	R.E. 3/10 L.E. H.M.	4/10 H.M.	40 60	20 30	Wide angle	R.E., cyclodialysis; L.E., Elliot operation (to compare)
22 J. C. M	85	Simple chronic glaucoma	R.E. Light L.E. 3/10	Light 4/10	28 35	28 20	Wide angle L.E.	R.E. operated with poor results (trephining). L.E., postoperative hyphema; the R.E. was not operated
23 J. H. M	50	Simple chronic glaucoma	R.E. 1/10 L.E. 9/10	4/10 9/10	60 25	22 25	Wide angle; corneal edema does not permit good observation	The considerable improvement obtained must be attributed to the disappearance of the corneal edema; only the R.E. was operated
24 N. B. M	68	Simple chronic glaucoma	R.E. H.M. L.E. 5/10	H.M., 7/10	30 30	30 15	Wide angle	Postoperative synechias, small tearing of Descemet's membrane. R.E. was not operated
25 F. D. T. M	70	R.E. simple chronic glaucoma L.E. irritative chronic glaucoma	R.E. 20/40 L.E. 0	20/40 0	60 80	20 80	R.E., wide angle L.E. cannot be observed	Sclerecto-cyclodialysis in both eyes: L.E., hyphema treated and improved with injections of sodium iodide and placenta implantation
26 D. C. F	69	Simple chronic glaucoma	R.E. 1/10 L.E. 1/10	1/10 1/10	30 40	15 30	Wide angle; incipient capsular exfoliation, embryotoxon	Operated on both eyes in the same session
27 M	6	Buphthalmos			45 20	15 20	—	L.E. operated by me when he was a year old with excellent results (trephining). Later a colleague, who cared for him, did not operate the R.E., thus giving origin to extensive buphthalmos. After operation, the corneal edema disappeared and the eyeball diminished in its size

This fact would seem to indicate an early operation but it cannot be used as an argument against the excellence of the procedure as a hypotonic operation. It was observed that in some cases (Cases 1 and 26) hypotony was rather slow in developing.

Hyphema during the operation or in the first postoperative days occurred in five cases (Cases 2, 10, 22, 25, and 27). In Case 10 peripheral synechias following cataract operation explained the extensive hyphema.

Diplopia occurred in two cases; in one of them the operation was performed through the external rectus separating the muscular bundles with two forceps. This method was discarded because it was more complicated.

Hernia of the vitreous occurred in four cases (Cases 4, 6, 23, and 25) but it did not result in any loss of vision.

Juncal 1330.

References

1. Heine: Augenarztliche Operationslehre. Berlin, 1922, v. 1, p. 872.
2. Elschnig: Ztschr. f. Augenh., **22**:365, 1909.
3. Stallard, H. B.: Eye Surgery. Baltimore, Williams & Wilkins, 1946, p. 306.
4. Blaskovicz and Kreiker: Eingriffe am Auge. Stuttgart, 1938, p. 343.
5. Gát: Ophthalmologica, **114**:107, 1947.
6. Campos: IV Congreso Brasileiro de Oftalmologia, Rio de Janeiro, 1941, v. 1, p. 319.
7. Danielson, Long, and Sherwood: Am. J. Ophth., **25**:454, 1942.
8. Randolph: Am. J. Ophth., **26**:187, 1943.
9. Mauksch: Ztschr. f. Augenh., **67**:313, 1929.
10. Wooton, Tr. Am. Ophth. Soc., **30**:64, 1932.
11. Wheeler: Arch. Ophth., **16**:569, 1936.
12. Del Barrio: Ann. d'ocul., **171**:977, 1934.
13. Cornet: Ann. d'ocul., **175**:678, 1938.
14. Queiroga: Iv Congreso Brasileiro de Oftalmologia, Rio de Janeiro, 1941, v. 1, p. 350.
15. Troncoso: Arch. Ophth., **23**:270, 1940.
16. Römer: In Blaskovicz and Kreiker: Eingriffe am Auge, Stuttgart, 1938, p. 277.
17. Sallmann: In Arruga: Cirugía Ocular, Barcelona, 1946, p. 679.
18. Mügge: Klin. Monatsbl. f. Augenh., **112**:104, 1947.
19. O'Brien and Weih: Arch. Ophth., **42**:606, 1949.
20. Lee and Allen: Am. J. Ophth., **32**:1713, 1949.
21. Sugar: Am. J. Ophth., **30**:843, 1947.
22. Shaffer: Am. J. Ophth., **30**:860, 1949.
23. Barkan: Am. J. Ophth., **30**:1063, 1947.
24. Salus: Klin. Monatsbl. f. Augenh., **64**:433, 1920.
25. Stein: Arch. f. Augenh., **102**:626, 1930.
26. Bunge: Klin. Monatsbl. f. Augenh., **90**:21, 1933.
27. Gradle: Am. J. Ophth., **3**:1, 1920.
28. Loddoni: Ann. di ottal, e clin. ocul., **54**:600, 1926.
29. Stoutenborough: Am. J. Ophth., **10**:260, 1927.
30. Wernicke: Klin. Monatsbl. f. Augenh., **46**:579, 1908.
31. Vannas: Klin. Monatsbl. f. Augenh., **95**:692, 1935.
32. Barkan, Boyle, and Maisler: Am. J. Ophth., **19**:21, 1936.
33. Clarke: Am. J. Ophth., **24**:788, 1941.
34. Lindner: In Blaskovica and Kreiker: Eingriffe am Auge, Stuttgart, 1938, p. 346.
35. Chandler: Arch. Ophth., **32**:23, 1944.

PERFORATING INJURIES OF THE GLOBE*

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This report is based on a statistical analysis† of 444 patients with perforating ocular injuries admitted to the Wilmer Institute during the years 1943 to 1951, inclusive. Completely excluded from this study were all injuries which were nonperforating, which had prior definitive treatment elsewhere, or in which initial examination was postponed so long that the outcome was predetermined before observation at the hospital.

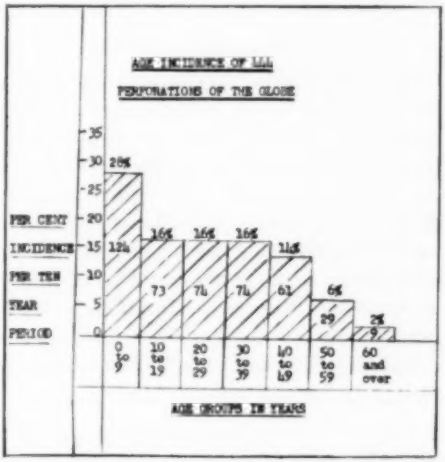
The distribution with respect to age, sex, race, and particular eye involved was studied in all 444 patients. The age distribution is shown in Graph 1. It can be seen that perforating ocular injuries occurred during the first decade of life almost twice as frequently as during any subsequent decade. Males outnumbered females four to one. The incidence in the white race was 63 percent, and in the colored race was 37 percent. This disproportion probably reflects the preponderance of the white population over the colored population in this hospital. The right eye and the left eye were each involved with equal frequency.

VISUAL ACUITY ACHIEVED

The final resultant visual acuity was studied only in those patients who had a minimum three months' follow-up‡ and in those whose eyes were enucleated prior to that time. The visual results in ocular perforations with intraocular foreign bodies were found to be the same as those with double perforating foreign bodies, and so these two groups were combined here.

Table 1 compares the visual results of 249 simple* perforating injuries with 60 perforating injuries complicated by intraocular or double perforating foreign bodies. The presence of an intraocular or double perforating foreign body per se was not found to reduce the visual prognosis below that of a simple perforating injury.

Graph 2 illustrates the visual prognosis in simple ocular perforations when immediate enucleation was not mandatory.



Graph 1 (Duke and Schimek). Age distribution in perforating injuries of the globe.

TABLE 1

FINAL VISUAL ACUITY OF SIMPLE PERFORATIONS AND FOREIGN-BODY PERFORATIONS

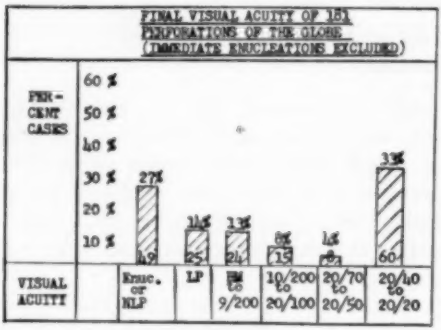
Final Visual Acuity	Enucl., NLP, & LP only	H.M. to 20/100	20/70 to 20/20
Simple perforating injuries (249 cases)	57%	16%	27%
Intraocular and double perforating foreign-body group (60 cases)	55%	20%	25%

* The word "simple" is used here to denote an ocular perforation not complicated by intraocular or double perforating foreign body.

* From the Wilmer Ophthalmological Institute of the Johns Hopkins Hospital and University.

† These findings were reported in more detailed form at the Wilmer Resident's Meeting, Baltimore, April 4, 1952.

‡ The average follow-up period of such cases was 27 months.



Graph 2 (Duke and Schimek). Visual prognosis in simple ocular perforations when immediate enucleation was not mandatory.

Initial complications

The important initial complicating factors were uveal prolapse, lens damage, and vitreous loss. Table 2 shows the incidence of these complications in simple perforations and in perforations with intraocular or double perforating foreign body. One striking finding was that uveal prolapse was almost seven times more frequent in simple perforations than in perforations complicated by foreign bodies. The most frequent complication in perforations complicated by foreign bodies was lens damage.

Factors influencing final vision in simple perforations

Final visual acuity was found to be most adversely influenced by lens damage, and to a lesser extent by increasing length of laceration. In analyzing those cases with lens damage, only perforations confined to the cornea were studied. This gave the cases under comparison an equal handicap with regard to corneal scarring. These analyses are shown in Table 3. In other similar analyses, neither the presence of uveal prolapse nor the age of the patient influenced the final visual result.

Table 4 shows that better visual results occurred if the injuries were caused by sharp edged agents as compared with blunt agents. Gunshot injuries had the worst prognosis of all.

The chi square method of analysis was employed to verify the above conclusions and also those to follow which pertain to infection.

Infection

Fifty-four percent of all cases were treated within six hours after the injury. The distribution of cases by interval of time between injury and initial treatment and the percent of eyes† in each time period which initially showed or developed infec-

† The eye was considered infected if it showed clinical or pathologic evidence of purulent inflammation within the eye.

TABLE 2

Incidence of initial complications in simple and foreign-body perforations

Initial Complications	Uveal Prolapse	Lens Damage	Vitreous Loss
Simple perforations (249 cases)	68%	43%	18%
Foreign-body perforations (60 cases)	10%	46%	11%

TABLE 3

Lens damage and length of laceration related to final vision

Type of Ocular Perforation	Total Cases	Visual Acuity Achieved			
		NPL	LP–9/200	10/200–20/100	20/70–20/20
With lens damage	65	17%	46%	14%	23%
Without lens damage	37	8%	3%	13%	76%
Laceration of 1 to 6 mm.	88	9%	26%	15%	50%
Laceration of 7+ mm.	59	15%	46%	10%	29%

TABLE 4

OUTCOME ACCORDING TO THE AGENT CAUSING THE INJURY

Agent	No.	NLP or Enuc.	LP to 5/200	10/200 to 20/100	20/70 to 20/50	20/40 to 20/20	20/70 or Better
Sharp metallic edge	59	28	16	2	2	11	22%
Glass	85	30	15	5	6	29	34%
Wood	15	4	4	5	2		13%
Blunt	64	42	10	5	1	6	11%
Miscellaneous	23	15	4		1	3	17%
Gunshot	11	8	3				0%

Note: Gunshot are the only intraocular foreign bodies included in above survey.

TABLE 5

OCCURRENCE OF INFECTION RELATED TO TIME INTERVAL BETWEEN INJURY AND TREATMENT

Time intervals in hours	0–1	2–6	7–12	13–24	25–48	48+	Unknown	Total
Total cases in each time interval	96	144	36	52	33	53	30	444
Infected cases in each time interval	5	11	8	7	8	8	1	48
% of infected cases in each time interval	5%	7%	22%	13%	24%	15%		

tion is shown in Table 5. There was a significant increase in the incidence of infection as the time period between injury and initial treatment lengthened.

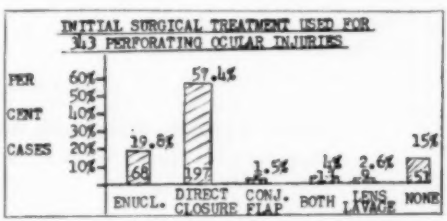
Of eyes with lens damage, 14.3 percent showed infection, whereas only eight percent of those without lens damage showed infection; 14.9 percent of perforations complicated by foreign bodies showed infection, whereas only 9.6 percent of simple perforations showed infection. Thus, the presence of a foreign body or lens damage was associated with a significantly higher incidence of infection. The presence or absence of uveal prolapse did not influence the incidence of infection.

It has been stated by Snell[1] in a previous study of this nature from the Wilmer Institute, covering the years 1935 to 1942, that "the sulfonamides, while probably of great value in a few cases, are on the whole unsatisfactory in the treatment of intraocular infections complicating penetrating ocular injuries."

In the present study, the great majority of eyes with intraocular infection resulted either in enucleation or in a very low visual acuity. Those few cases adequately treated with penicillin did not achieve significantly better results.

TREATMENT EMPLOYED

Graph 3 shows the relative frequency of the initial operative procedures employed for simple perforating ocular injuries. From a similar series of cases during the years 1935 to 1942 inclusive, reported by Snell, the following comparisons may be made. There was a similar incidence of cases which did not require surgery; thus the criteria for the



Graph 3 (Duke and Schimek). Relative frequency of initial operative procedures in simple perforating ocular injuries.

TABLE 6

PRINCIPAL CAUSAL FACTORS FOR ENUCLEATION

Factor	Number	Percent
Irreparable prolapse of intraocular contents	73	50.6
Infection	20	13.9
Phthisis	19	13.2
Persistent or repeated hyphema	13	9
Glaucoma	4	2.7
Sympathetic ophthalmia	3	2.1
Phaco-anaphylaxis	2	1.4
Miscellaneous	10	6.9
Total	144	

use of the pressure dressing have remained the same.

The conjunctival flap was employed 20 times more frequently in Snell's series than in the present series. This reflects the recent increased use of direct closure with fine sutures at the Wilmer Institute. It is of interest to point out that the over-all visual results of Snell's series were exactly the same as the present series—one quarter of the cases resulted in enucleation or no light perception; one third achieved a visual acuity of 20/40 to 20/20—despite the decreased use of the conjunctival-flap operation, the increased use of direct closure with finer sutures, and the increased use of modern antibiotics.

LATE COMPLICATIONS

The relative frequency of the causal factors for enucleation is shown in Table 6. The relative frequency of causal factors for reduction of final visual acuity is shown in Table 7. These tables are self-explanatory.

There were three clear-cut cases of sympathetic ophthalmia among 208 cases which had a minimum three-month follow-up period or in which the eyes were enucleated after the ninth day following injury. In two of these cases, sympathetic ophthalmia was proven by pathologic examination. The incidence of sympathetic ophthalmia based on the three clear-cut cases was 1.4 percent. In addition, there was one probable case, and if this is included, the incidence becomes 1.9 percent.

TABLE 7

PRIME CAUSAL FACTORS FOR REDUCTION OF FINAL VISUAL ACUITY TO 20/40 OR LESS (116 CASES)

Factor	Number	Percent
Lens damage	48	41.3
Phthisis bulbi	24	20.6
Corneal scar	22	18.9
Vitreous opacities	11	9.4
Retinal damage	4	3.4
Miscellaneous	7	6

SUMMARY

In summary, this has been an analysis of 444 perforating injuries of the globe with respect to incidence, complications, treatment, and final visual results.

The Johns Hopkins Hospital (5).

REFERENCES

1. Snell, A. C., Jr.: Perforating ocular injuries. Am. J. Ophth., **28**:263, 1945.

OPHTHALMIC MINIATURE

Then I went and visited poor Will Congreve, who had a French fellow tampering with one of his eyes: he is almost blind of both.

Swift, *Journal to Stella, Jan.* 6, 1711-12.

MONILIASIS*

A review and a report of the first case demonstrating the Candida albicans in the cornea

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Moniliasis is an acute or subacute infection in which the fungus producing this disease is caused by the species Candida, usually Candida albicans. The lesions may involve the eyes, mouth, vagina, skin, nails, and lungs. It may also produce a septicemia, endocarditis, or meningitis.

Synonyms of species of Candida

The generic name Monilia now has been replaced by the name Candida. This group of organisms of Candida are mycelia-producing, nonascospore-forming, yeastlike fungi. In the literature there are over 170 synonyms for the single species C. albicans which is the pathogenic member of this group.

Hippocratic writers described thrush as a disease under the general term of aphthae. The nature of this disease was obscure. Langenbeck, Berg, and Gruby, in 1840, described deposits of the specific fungus of Monilia which is now called Candida, and at once the parasitic nature of the disease was established.

Robin, in 1853, called this fungus Oidium albicans. Zopf, in 1890, described the same fungus and called it Monilia albicans. Vuillemin, in 1898, called it Endomyces albicans since he found endospores within the mycelial filaments. Castellani and Chalmers, in 1913, called it Monilia pinoyi.

Ashford in 1917 made a study of cases in Puerto Rico. He isolated from the tongue and stools of 200 patients a fungus which he called Monilia psilosis.

Moniliasis has appeared in all parts of the world. The fungus occurs in healthy individuals in a variety of clinical forms.

Source of infection

Pathogenic strains of C. albicans can be isolated from (1) normal skin, (2) normal oral or vaginal mucous membranes, (3) stools of normal individuals, (4) canine genital region,[1] and (5) normal conjunctiva. Most infections are endogenous, but the determination of the source of infection is a difficult problem just as it is with Staphylococcus aureus infections.

The infection may be contagious and true epidemics have occurred. Balanoposthitis has been observed in the husbands of women with C. albicans vaginitis, and cutaneous moniliasis has occurred around the nipples of mothers nursing infants suffering from oral thrush. Epidemics of thrush have been noted in infants.

Moniliasis has been seen in fruit packers and in dish washers. When the skin is macerated and repeatedly placed in water this type of infection can occur.

In the eyes, the source of infection is usually endogenous but it can be exogenous. In 1938, Fazakas had demonstrated four instances of Candida from 1,791 eyes that were cultured.

Moniliasis is seen in all ages, races, and in both sexes. Lesions of hands occur in handlers of food and fruit packers. Lesions of the mouth are seen in infants as thrush, and in adults with artificial teeth. Pregnancy and diabetes are predisposing factors for Monilia vaginitis. Monilia endocarditis have been seen in drug addicts.

Signs and Symptoms

In the mouth the lesions appear as creamy white patches. The base of the patch is pink and moist. Moniliasis of the tongue is a hairy form of glossitis which is called "hairy

* From the Veterans Administration Center.

tongue." Perleche is seen as cracks or fissures in the corners of the mouth.

In Monilia vulvovaginitis the lesion can be seen as eczematoid dermatitis, or excoriated vesicular pustules or ulcers may occur.

Three forms of cutaneous lesions are seen: (1) Localized lesions, (2) generalized lesions, and (3) moniliids. Obesity and excessive sweating are factors that produce these cutaneous lesions, as well as diabetes and unhygienic surroundings.

Some of the localized lesions are onychia and paronychia, intertrigo and perianal moniliasis.

Generalized cutaneous lesions appear on the glabrous skin and may be in the form of an eczematoid type or associated with vesicles or pustules. These lesions are associated with glossitis or stomatitis.

The moniliids or "levurids" are sterile, grouped vesicular lesions and are found on the hands and body and also on the eyelids. They are due to an allergic manifestation of moniliasis elsewhere in the body.

Cough is an important symptom in the bronchial form of this infection. The physical signs are of bronchitis with coarse rales at the bases of the lungs. The diagnosis is made by examining the sputum for the budding fungus and also by X-ray pictures. The X-ray plates show peribronchial thickening or a hazy type of linear fibrosis.

The true pulmonary moniliasis is serious. It is associated with fever, rapid pulse, and pleural pain. Cough produces a thick sputum which contains blood. It may appear as a patchy or bronchopneumonia or as a lobar pneumonia with increased dullness and moist rales. Death may occur when two or more lobes are involved.

The X-ray findings vary in size and shape and may resemble bronchopneumonia except that the edges of the lesions are less sharply defined. The apices are spared but two or more lobes are involved. The films made at different intervals may show healing in some portions and spreading in other parts of the lung tissue. When the infection is severe, the lesion is massive, smooth, and dense and involves an entire lobe. Some lobes may show consolidation and other lobes only patches of consolidation.

Bone and joint infections have been reported.

Endocarditis seen in drug addicts resembles subacute bacterial endocarditis. The fungus isolated resembles C. parakrusei. Candida krusei[2] has also been reported from a case of mycotic endocarditis.

Ocular tissues and adnexa

1. *Eyelids.* Monilial blepharitis may be seen in infants who present generalized cutaneous lesions of the face. The lesions may be eczematoid, or the eyelids may be covered with vesicles and pustules similar to the lesions on the face proper. Ruiz,[3] in 1947, reported eczematoid moniliids or "levurids" of the eyelids.

2. *Conjunctiva.* Pichler, in 1900, reported two cases of monilial conjunctivitis. One showed the clinical picture of pseudomembranous conjunctivitis and the other case had a scabbed necrotic condition of conjunctiva and cornea, which was associated with a similar condition in the mouth and nose and resulted in shrinkage of conjunctiva and opacity of the cornea.

Norton[4] reported a case of conjunctivitis in 1927.

Duke-Elder[5] states that fungus infections of the conjunctiva are rare; they cause lesions of a granulomatous nature and the diagnosis rests entirely on identification of the organism.

Maddren,[6] in 1941, reported a case of angular conjunctivitis of extensive and long-standing cutaneous moniliasis in a woman.

3. *Cornea.* Pichler, as has been noted, reported a case of keratoconjunctivitis.

Yalour,[7] in 1935, reported a case of central serpiginous ulcer of each eye accompanied by small nodular buccal and pharyngeal lesions. From all the lesions Candida albicans was isolated and cultured.

I am reporting a case of corneal ulcer due

to C. albicans. This is the first case to be reported demonstrating this fungus in an ulcer involving all the layers of the cornea with pathologic illustrations of the enucleated eye. A complete microscopic description will be given elsewhere (fig. 2). This case of moniliasis of the cornea developed after a foreign body entered the patient's left eye.

I first examined this patient two weeks after his injury. His left eye presented a gray ulcer of the entire cornea. The ulcer was dry, resembled bread crumbs, and had the appearance of a dry scab of necrotic tissue. When the corneal scraping was performed, the base of the ulcer had the appearance of a pink, glistening membrane.

A marked hypopyon with an associated iritis was also present. The ulcer perforated one month following injury, which necessitated an enucleation of his left eye.

4. *Uvea, retina, and vitreous.* Miale[8] reported on an eye that was enucleated in 1937. The clinical impression was panophthalmitis on the right eye, probably tuberculosis. There was tuberculosis of the mouth. Sections of the eye were interpreted as showing an acute and chronic granulomatous lesion. All coats were involved. Acid-fast stains of sections were negative for tubercle bacilli.

The patient died in 1941 with a questionable tuberculosis of his meninges. The anatomic autopsy diagnosis was chronic stomatitis, glossitis, and laryngitis due to Candida albicans; meningitis due to C. albicans.

The right eye that had been enucleated in 1937 was now restudied with special stains which showed a large aggregate of typical mycelia in the exudate and necrotic tissue obliterating the posterior chamber and vitreous. The brain sections also showed mycelia of C. albicans.

Miale stresses that mycosis should be considered in all cases of nonspecific granuloma. In these cases Gram stain identifies the fungus in tissue section. He also states that the yeast cells can be mistaken for small lymphocytes.

The case herein reported shows evidence of iritis with detachment of the ciliary body, choroid, and retina. Hemorrhages were noted in the retina and vitreous (fig. 4).

Laboratory diagnosis

Direct microscopic examination

Skin and nail scrapings should be placed in a drop of 10- to 20-percent potassium hydroxide, a cover slip added, and the preparation cleared by gently heating over a flame. Sputum, mucus, and other material not requiring preliminary clearing should be flattened to a thin film on a slide under a cover slip. The fungus in these preparations appears as oval, budding, thin-walled, yeast-like cells measuring from two to four microns. Occasionally, pseudomycelia, with budding cells attached to the points of constriction, are noted. The fungus is stained by the method of Gram; both cells and pseudomycelia are gram-positive.

Culture

Material should be cultured on Sabouraud's glucose-agar slants and grown at room and incubator temperatures. Growth occurs in from two to five days. Colonies are medium sized, soft, creamy, and moist to dull in appearance. Cultures have a characteristic yeasty odor.

Differentiation

Candida albicans does not grow on the surface of Sabouraud's broth, produces acid and gas from glucose and maltose acid from sucrose, and fails to attack lactose. On corn-meal agar at room temperature, typical chlamydospores are produced.

This latter characteristic is perhaps the simplest and most rapid method of differentiating Candida albicans from the other nonpathogenic Candida species. Several cuts are made in the surface of a plate of corn-meal agar with a platinum wire inoculated with the fungus. On this medium, Candida albicans produces clusters of cells bearing pseudomycelia and the round, thick-walled chlamydospores (fig. 1).

Commercially manufactured and some kinds of homemade corn-meal agar are not satisfactory. This laboratory obtains consistently good results with the medium made according to the following formula: Yellow corn meal, 40 gm.; agar, 18 gm.; distilled water, 1,000 cc. Simmer corn meal in water for one hour, filter through gauze and make up to original volume, add agar and melt in autoclave. Filter through cotton and gauze, tube, and sterilize at 15 pounds for 15 minutes.

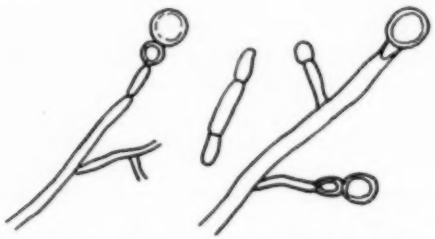

Fig. 1 (Mendelblatt). Characteristic terminal chlamydospores of Candida albicans on corn-meal agar.

Animal inoculation

Since Candida albicans is the only pathogenic member of the group, it may be identified on the basis of its pathogenicity for animals. One cc. of a one-percent saline suspension injected intravenously into a rabbit results in death of the animal in four or five days. Autopsy reveals numerous small, white, cortical abscesses in the kidneys which show the fungus in direct smears.

Table 1 summarizes the differential diagnosis of species of Candida.

Histopathology

Biopsies may show a granulomatous lesion with giant cells and epithelioid cells. This may be confused with tuberculosis. Necrotic areas may be present and the epithelium may be ulcerated, with round cells noted.

The fungus is gram-positive and will be present in the necrotic tissue. The mycelia are slender rods and contain blastospores.

TABLE 1

Differential diagnosis of species of Candida
(From Martin and others, 1937)

Medium	Pathogenic	Nonpathogenic					
	C. albicans	C. tropicalis	C. pseudotropicalis	C. Krusei	C. parakrusei	C. stellatoidea	C. guilliermondi
Sabouraud's agar Sabouraud's broth	Creamy growth No surface growth	Not characteristic Narrow surface film with bubbles	Not characteristic No surface growth	Flat, dry Wide surface film	Creamy No surface growth	Creamy No surface growth	Creamy No surface growth
Blood agar	Medium-sized, dull-gray colonies	Large, gray colonies rounded by mycelial fringe	Colonies small, not characteristic	Colonies small, irregularly shaped, flat or heaped	Colonies small, brilliant white	Colonies star-shaped	Medium-sized dull-gray colonies
Corn meal	Branched, treelike mycelium with chlamydospores	Mycelium well-developed, branched, bearing numerous blastospores, no chlamydospores	Mycelium poorly developed, no chlamydospores	"Crossed sticks" mycelium, no chlamydospores	Mycelium well developed, no chlamydospores	Mycelium with large, ball-like clusters of blastospores	Mycelium well-developed, no chlamydospores

For a definite diagnosis of moniliasis the biopsy material should be cultured.

In some cases the correct diagnosis is made only at autopsy. The autopsy section of the organs involved will show the blastospores of C. albicans. The mycelia should not be confused with any small round cells that are present.

Immunology

Agglutinins have been noted in serum of patients with moniliasis in titers as high as 1:2,400. Serology is not a valuable diagnostic aid.

A large percentage of normal patients give positive skin reactions when injected with vaccines of C. albicans. Although a positive or negative test has little diagnostic value, skin tests should be performed on all patients with moniliasis because the care of the patient depends on whether the patient is hypersensitive to the fungus.

Prognosis

Localized types of moniliasis usually respond to treatment. Generalized cutaneous forms, or hypersensitive patients with moniliasis, are resistant to treatment. In bronchial and pulmonary forms, the patient usually recovers, the pulmonary form may, however, be fatal. Cases of endocarditis or meningitis have a hopeless prognosis.

Treatment

The prophylactic treatment includes:

1. The patient should be placed in clean, hygienic surroundings.
2. Regulate the diabetic patient.
3. Reduce the weight if obesity is present.
4. Restore the patient's general resistance with a well-balanced diet and plenty of vitamins.
5. The patient should be instructed to sterilize all needles and syringes carefully because unsterile needles may also produce moniliasis. The oral lesions respond to alkaline mouth washes. They can also be painted with one- or two-percent gentian violet either in aqueous or alcoholic solution.

Topical therapy of cutaneous moniliasis depends on the location of the lesion. Where possible, solutions of potassium permanganate of 1:5,000 may be used as wet dressings and then the parts may be painted with one-percent gentian violet solution.

On the eyelids one- to three-percent ammoniated mercury ointments can be used. One-percent undecylenic acid in an ointment base can also be used topically on the eyelids. Cases which resist therapy can be treated with fractional X-ray therapy. The more resistant forms can be treated by vaccine desensitization with oidiomycin.

Bronchopulmonary moniliasis responds to potassium-iodide therapy. Pulmonary moniliasis can best be treated with intravenous gentian violet. Antibiotics have not proved successful in the treatment of moniliasis, either systemically or by topical application.

Case report

History. W. A., a 56-year-old white man, was admitted to the Veterans Administration Hospital, Bay Pines, Florida, on April 12, 1950. His chief complaint was a sensation of pain in his left eye.

The onset of his present illness dated to March 30, 1950, when he felt a foreign body enter his left eye. The next day he had a painful left eye and, over the week end, the pain became progressively more severe. He then consulted a physician and for two weeks his left eye did not respond to therapy. His physician told him that he had a severe ulcer of his left eye and he should be placed in a hospital for further therapy.

Physical examination. He was well-nourished. The general physical examination was negative with the exception of an absent right testicle, removed at the age of 12 years because of mumps.

Laboratory findings. Urinalysis and complete blood count were negative. Kahn was negative. Corneal scrapings of the left eye on admission showed Candida albicans by the Gram stain. Cultures of the corneal ulcer were negative for growth.

Fig. 2 (Mendelblatt). Candida albicans in the area of the corneal ulcer (×215).

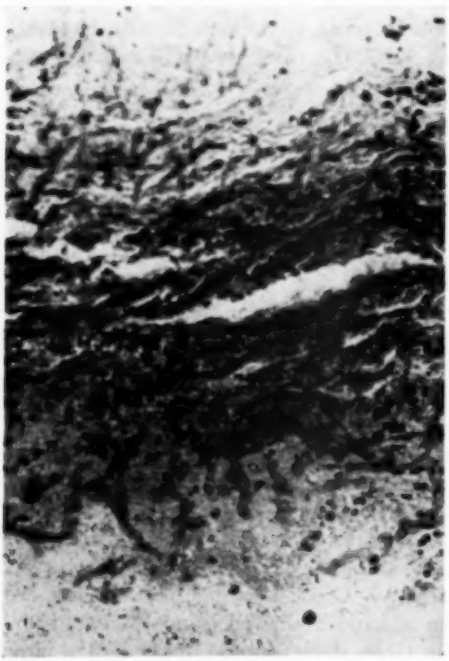

Fig. 3 (Mendelblatt). Candida albicans throughout the depth of the cornea (×280).

X-ray findings for heart and lungs were negative.

Ophthalmic examination. On admission the right eye showed essentially negative findings. Vision was: R.E., 20/30; L.E., 20/100, uncorrected. Vision in the left eye could not be corrected. The left eye had deep ciliary injection. The cornea had a gray ulcer, which was dry and had the appearance of bread crumbs, at the pupillary area. It resembled a dry scab of necrotic tissue. A marked hypopyon and an associated iritis were also present.

Hospital course. The ulcer was cauterized daily with iodine and every known antibiotic was used topically, but the patient did not respond to our therapy. The left eye became blind and painful. On April 29, 1950, the ulcer perforated and enucleation was performed on May 2, 1950. He was discharged May 12, 1950, with a well-fitted prosthesis.

Pathologic report

The enucleated eye was sent to the L. F. Montgomery Laboratory of Ocular Pathology, Emory University, for pathologic study. The following report was submitted by Dr. F. Phinizy Calhoun, Jr., director:

"*Microscopic.* There is a large perforated corneal ulcer in the temporal half of the cornea. The walls of the ulcer are dense masses of a yeastlike fungus and the coagulum in the ulcer crater contains many of the same organisms (figs. 2 and 3). There is also a cellular reaction, mainly polymorphonuclear with a few small round cells, about the ulcer.

"The fungi are seen throughout the depth of the cornea but none are definitely seen to penetrate into the anterior chamber. There is moderate episcleral infiltration of small round cells and vascular dilatation.

"A hypopyon is present. The hypopyon partially blocks the filtration angle on one side and a few pus cells are seen in the opposite filtration angle. Eosinophils in small numbers are present in the hypopyon.

"The iris and ciliary bodies are infiltrated with small round cells, polymorphonuclear

leukocytes, and eosinophils. The ciliary body is partially detached on both sides by pink-staining fluid. The lens is missing.

"The choroid is also partially detached by fluid. The choroidal vessels are sclerosed and dilated, especially posteriorly.

"The retina is detached over one small area by fluid, and there are several small collections of blood beneath the retina. Small and large hemorrhages are seen in all of the layers of the retina. Some of the blood has escaped into the vitreous. There is also some edema of the retina, especially posteriorly and in the outer plexiform layer (fig. 4). The optic disc is swollen.

"Diagnosis: Corneal ulcers. Monilia albicans (history)."

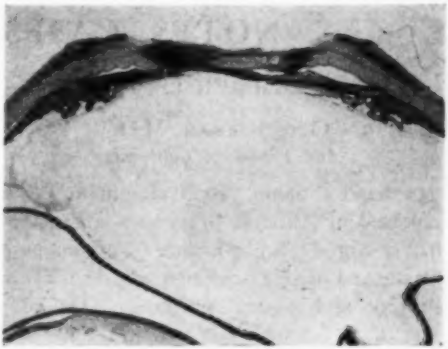

Fig. 4 (Mendelblatt). Detachment of the ciliary body, choroid, and retina (×8).

Summary

A case of Candida albicans of the cornea which resulted in perforation and finally enucleation of the eye is reported. This appears to be the first case in the literature demonstrating the fungus in the cornea and accompanied by a complete pathologic report. The literature on ocular moniliasis has been reviewed. Moniliasis of other organs of the body has been outlined and therapy discussed.

Moniliasis should be suspected in non-specific granulomas. It should not be confused with tuberculosis. In microscopic sections of biopsy or autopsy material, the Candida albicans should not be confused with small lymphocytes.

I wish to express appreciation to the W. B. Saunders Company, Philadelphia, for permission to use material from the *Manual of Clinical Mycology*. I also wish to thank Dr. Elbert DeCoursey, director, Armed Forces Institute of Pathology, for making the photomicrographs; Dr. F. Phinizy Calhoun, Jr., for the pathologic report; Mr. Charles Hillier, bacteriologist, Veterans Administration Hospital, Bay Pines, Florida, for his illustration of Candida albicans and for confirming the bacteriologic diagnosis from the corneal scrapings.

References

1. Reich, W. J., and Nechtow, M. J.: Canine genital moniliasis as a source of re-infection in human female. J.A.M.A., **141**:991 (Dec.) 1949.
2. Wolee, E. I., and Henderson, W.: Mycotic endocarditis. J.A.M.A., **147**:1344, 1951.
3. Ruiz, M. G.: Eczematoid moniliid of eyelids ("Candida"). Allergy, **5**:132-36 (Mar.-Apr.) 1947.
4. Norton, A. H.: Thrush of conjunctiva. Am. J. Ophth., **10**:357, 1927.
5. Duke-Elder, W. S.: Textbook of Ophthalmology. St. Louis, Mosby, 1938, v. 2, p. 1654.
6. Maddren, R. F.: Conjunctivitis, Am. J. Ophth. **24**:307 (Nov.) 1941.
7. Yalour, R. R., Negri, T., and Balza, J. F.: Blastomycotic corneal ulcer. Arch. de Oftal. de Buenos Aires, **10**:78, 1935.
8. Miale, J. B.: Candida albicans infection confused with tuberculosis. Arch. Path., **35**:427-437 (Mar.) 1943.

Ophthalmic Miniature

I like to operate about 2:00 p.m. so that the patient can have lunch and a glass of wine before hand.

Henry Power, *Diseases of the Eye*, 1860.

NOTES, CASES, INSTRUMENTS

A HAMMER LAMP*

OTTO BARKAN, M.D.
San Francisco, California

A hand slitlamp (focal illuminator) was reported in 1946.† It proved so satisfactory that a larger lamp or hammer lamp made of plastic and of similar design was devised for surgery and general purposes. This lamp (fig. 1) is 5.5 inches in length, weighs 10.5 ounces, and furnishes brilliant illumination, free from color.

Fig. 1

Fig. 2

Figs. 1 and 2 (Barkan). (1) Hammer lamp. (2) Hammer lamp attached to plastic rod.

The light source is a bulb operating on eight volts, the transformer being a free unit when the lamp is used with a handle or built into the base of the flexible goose-neck stand when used in this manner. Various accessories are made for it, such as transilluminators and suitable filters.

The light weight, relatively small size, and comparative freedom from heat of this lamp, combined with its illuminating powers, have made it particularly satisfactory for use in the operating room, as well as in the office.

For surgery, the lamp may be attached to a plastic rod four feet long which affords facility and ease in holding and positioning.

490 Post Street (2).

* These lamps may be obtained from Parsons & Company, 518 Powell Street, San Francisco, California.

† Barkan, Otto: A new focal illuminator. Am. J. Ophth. **24**:439 (April) 1941.

PHOTOGRAPHY OF THE EXTERNAL EYE

A SIMPLE, INEXPENSIVE TECHNIQUE

IRA A. ABRAHAMSON, JR., LIEUT. (MC) U.S.A.
Camp Gordon, Georgia

Photographic reproduction of the anterior segment of the eye has become a practical and efficient aid to the ophthalmologist. It is one of the best methods for recording clinical ocular pathology both for research, teaching, and medicolegal illustrations.

The method to be described is especially adaptable for taking pictures, of pre- and postoperative squint cases, enucleations, implants, cataracts, lid cases, and so forth. The advocated technique provides stability of the apparatus and patient, accuracy of aim, good depth of focus, inexpensive, nonirritating illumination, and a rapid, easy, efficient mode of operation.

Many articles have appeared on this subject in the past 12 years. Those by Knighton,[1] Bedell,[2] Bogart,[3] Irvine,[4] Katzin,[5] Landers,[6] Callahan,[7] Sysi,[8] Knight,[9] Hansell,[10] Douvas,[11] Chace,[12] and Donaldson,[13] have been particularly instructive. Some of the methods described are quite good but most are too expensive or complicated, with handicaps of instability and lighting disadvantages.

The following is a description of the apparatus used.

APPARATUS (Figs. 1 and 2)

1. *Slitlamp table with chinrest.* Almost every eye clinic or ophthalmologist's office has a slitlamp. The type used by me is the

Bausch and Lomb universal slitlamp table. The chinrest aids in stabilizing the patient and can be elevated and depressed to the desired height to maintain alignment. It should be noted that the apparatus (fig. 2) herein described does not have to be fastened to the slitlamp table, but may be used on the wards or in the operating room with the only defect being instability.

Block of wood. This is 7¾ by 3½ by 3¾ inches in dimension. It is screwed into the under surface of the slitlamp table and left permanently in place to support the arm.

2. *Arm.* This is made out of a broomstick handle and is 12 inches long. There is a screw two inches long inserted into one end of it (a). This fastens into the block of wood, described above, projecting from the under edge of the slitlamp table. It is held firm by a fly wing bolt (b).

3. *Light cross bar.* This contains the light source and supports the camera. It fastens to the upper end of the arm and is left permanently in place. The type used is a Multi-Lite, Jr., manufactured by Mayfair Company, Brooklyn, New York.

Light source. Two 100-watt, 120-volt bulbs are used. They are inexpensive, non-irritating to the eye, and provide adequate illumination.

4. *Camera.* An Argus C-3, Leica, Contax, or any type may be used.

Adapter. This fits over the lens mount and supports the portrait lens.

A portrait lens fits over the camera lens and is essential for close-up photography. A plus 3.0D. meniscus lens may be used for objects 11 and one-half inches away.

Extension cable. This aids in stabilizing the camera.

Film. The camera is always loaded, ready for use, with either black and white Kodak Panatomic-X or Kodachrome Indoor Type A 35-mm. film.

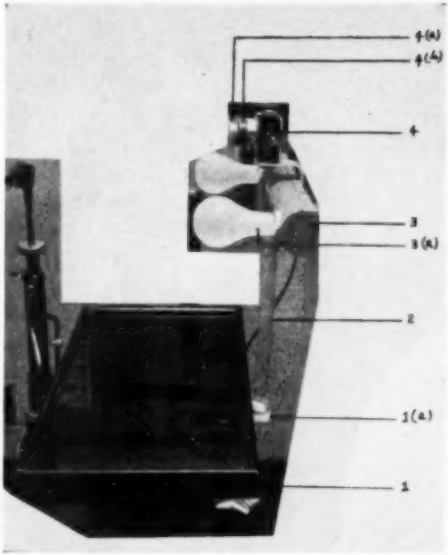

Fig. 1 (Abrahamson). Slitlamp table and apparatus.

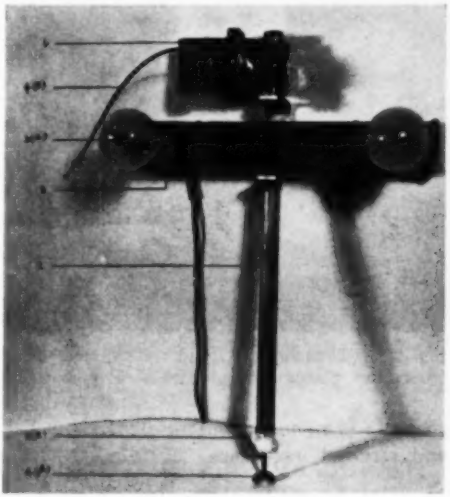

Fig. 2 (Abrahamson). Apparatus alone.

Technique

The patient is seated facing the apparatus with his chin on the chin rest. Figure 3 demonstrates the operation of the apparatus. The arm (2) and cross bar (3), which are permanently attached, are fastened into place on the block of wood (1a).

The camera (4), whose speed is constant on "bulb" and whose lens aperture is fixed

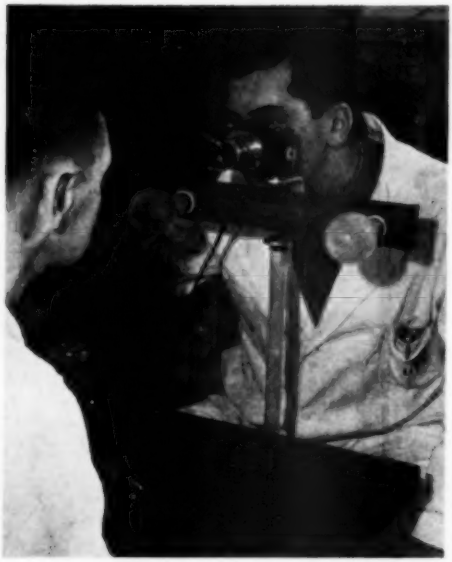

Fig. 3 (Abrahamson). Photographic technique.

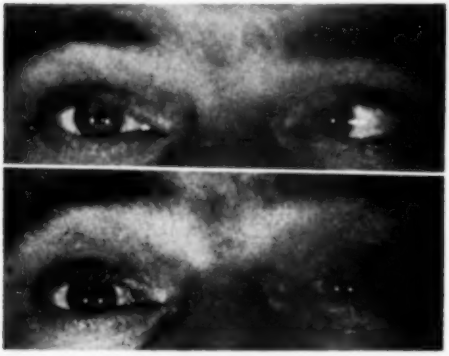

Fig. 4 (Abrahamson). Preoperative and postoperative photographs in a case of esotropia.

at F4.5, is mounted on the light cross bar and screwed into place.

The lens is lined up with the bridge of the patient's nose by adjusting the chinrest. The patient's eyes are under constant observation for blinking and, in a matter of seconds, the picture is taken. Figures 4 and 5 show the size of field and depth of focus obtained.

Only a few minutes are required to mount the apparatus on the slitlamp table and take the picture. The technique is so simple, that a nurse, corpsman, or secretary can operate it easily.

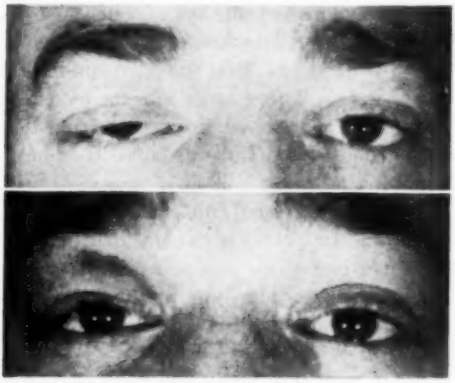

Fig. 5 (Abrahamson). Preoperative and postoperative photographs in a case of ptosis.

Summary

A simple, inexpensive technique for photographically reproducing the anterior segment of the eye is described. The method provides stability of the apparatus, accuracy of aim, good depth of focus, inexpensive, nonirritating illumination, and a rapid, easy, efficient mode of operation.

EENT Clinic, U. S. Army Hospital.

References

1. Knighton, W. S.: A simple set-up for external eye photography. Am. J. Ophth., **21**:300 (Mar.) 1938.
2. Bedell, A. J.: Colour photography in Ophthalmology: In Ridley, F., and Sorsby, A.: Modern Trends in Ophthalmology. London, Butterworth, 1940, pp. 213-233.
3. Bogart, D. W.: Clinical photography with special reference to photography of the anterior segment of the eye. Am. J. Ophth., **25**:61-66, (Jan.) 1942.
4. Irvine, R., and Stimson, R.: A method of ultra close up photography in ophthalmology. Arch. Ophth., **23**:161-163 (Jan.) 1940.
5. Katzin, H. M.: Photography of the external eye. Arch Ophth., **35**:514-518, 1946.
6. Landers, P. H.: Simplified external eye photography. Am. J. Ophth., **31**:1624-1626, 1948.

7. Callahan, A.: Photography of the eye. Med. Radiog. 24:46-53, 1948.
8. Sysi, R.: Simple device supplementary to slitlamp to photograph parts of eye. Acta Ophth., 27:403-408, 1949.
9. Wright, E. S.: Survey of anterior segment photography. Am. J. Ophth., 32:1245-1251, 1949.
10. Hansell, P., and others: Discussion of ophthalmic photography. Proc. Roy. Soc. Med. (London), 42:451-458, 1949.
11. Douvas, N., and Allen, L.: Anterior segment photography. With Nordenson retinal camera. Am. J. Ophth., 33:291-292, 1950.
12. Chace, R., and Lafayette, J.: A lighting system for photography of the eye. Arch. Ophth., 43:371-373, 1950.
13. Donaldson, D.: Camera for sterioscopic photography of anterior segment. Arch. Ophth., 43:1083-1087, 1950.

SENSITIVITY TESTS OF STAPHYLOCOCCUS CULTURES*

USING 30-PERCENT SODIUM SULFACETIMIDE

HORACE W. SHRECK, LIEUT. COL. (MC) U.S.A.
Ancon, Canal Zone

In conjunction with a clinical study of sulfacetimide in staphylococcal infections, I have made a laboratory survey of sensitivity tests on staphylococcal cultures with Dr. Joel Shrager, chief of the bacteriology section, Board of Health Laboratory, Gorgas Hospital. Two hundred staphylococcal cultures were tested for sulfacetimide sensitivity. The tests were performed with sodium sulfacetimide, using discs saturated with a 30-percent solution, which was also applied to blood-agar plates that had been plated with the staphylococcus being checked. The cup technique was also used in which the 30-percent sulfacetimide was in direct contact with the media. In all instances one method checked the other. Both coagulase-positive and coagulase-negative cultures were used.

In the 200 staphylococcal cultures the following types were seen:

Staphylococcus albus—nine, of which one was coagulase positive and eight were coagulase negative.

Staphylococcus albus-hemolyticus—five, of which one was coagulase positive and four were coagulase negative.

Staphylococcus aureus—11, of which three were coagulase positive and eight were coagulase negative.

Staphylococcus hemolyticus—175, of which 51 were coagulase positive and 124 coagulase negative.

Of the 200 cultures, 18 were sensitive to sulfacetimide (30 percent); of the 18, three were coagulase positive and 15 were coagulase negative.

The results of these sensitivity tests, which followed rather closely those of our clinical study, were disappointing; 30-percent sulfacetimide had given better clinical results in the states.

Dr. Joel Shrager, the chief of bacteriology, Board of Health Laboratory at Gorgas Hospital (whom I wish to thank for invaluable assistance), with whom I discussed possible explanations for the lack of sensitivity, felt that definitely different strains of staphylococcal organisms are present in the Canal Zone. He felt that the climate, humidity, and so forth did not affect the organisms and that they were not factors to be considered.

Gorgas Hospital.

* From the Department of Ophthalmology, Gorgas Hospital.

ACUTE GLAUCOMA DURING CORTISONE THERAPY

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Utica, New York

The response of secondary glaucoma in uveitis to systemic cortisone or ACTH treatment seems to be unpredictable, some cases actually react with an increase of intra-

ocular pressure. Secondary glaucoma may even develop under cortisone or ACTH treatment.[1] Woods[2] also noted inconsistent results in secondary glaucoma. In primary glaucoma, ACTH does not have any effect on the intraocular pressure.[3]

Tillet[4] studied the effect of ACTH on the intraocular pressure of normal eyes in 15 patients who received it for nonocular conditions and stated that he had found no significant elevation during treatment; in 10 patients under active ACTH treatment, the response to provocative tests for glaucoma was negative. It may be that this worker was a little too cautious in the evaluation of his results; five of his patients actually showed an increase of three or four mm. Hg in one or both eyes after ACTH treatment was started.

The following case is presented even though it is realized that the occurrence of congestive glaucoma during systemic cortisone treatment may have been entirely coincidental. There are one or two points, however, which may be significant.

Case report

A woman, aged 59 years, had been complaining of a bad cold and her family physician had given her a penicillin injection. She responded with a violent allergic reaction of face, chest, and hands. This was treated with oral cortisone—200 mg. the first day, 100 the second, and 50 for two more days.

On the fourth day, she started to complain of pain and blurred vision in the left eye and, two days later, when she was first seen by me, there was an acute congestive glaucoma present in the left eye. Tension was 45 mm. Hg (Schiøtz) and vision was reduced to perception of hand movements.

Energetic treatment with pilocarpine and eserine was instigated and, within eight hours, the tension had become normal. After three days the eye had settled down, vision was restored, the field complete, and the tension was equal and normal in both eyes. After three weeks, all treatment was discontinued and tension, vision, and fields have remained unchanged to the date of writing, eight months after the attack.

It is impossible to decide whether the glaucoma attack was precipitated by the cortisone treatment in an eye predisposed to glaucoma, perhaps on the basis of water retention (the patient was not on a salt-restricted diet). There are some points in favor of this assumption: The complete absence of prodromal signs previous to the first attack, the immediate effect of a short routine treatment, and the absence of any further signs of glaucoma.

3 Hopper Street.

References

1. McLean, J. M.: Discussion of paper by Woods, A. C.: Use of ACTH and cortisone. Tr. Am. Ophth. Soc., **48**:259-296, 1950.

2. Woods, A. C.: The present status of ACTH and cortisone in clinical ophthalmology. Am. J. Ophth., **34**:945-960 (July) 1951.

3. Blake, E. M., Fasanella, R. M., and Wong, A. S.: The effect of ACTH in glaucoma. Am. J. Ophth., **33**:1231-1235 (Aug.) 1950.

4. Tillet, C. W.: Effect of ACTH on normal ocular tension. Am. J. Ophth., **35**:659-662 (May) 1952.

SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

YALE UNIVERSITY CLINICAL CONFERENCES

October 10, 1952

DR. R. M. FASANELLA, *presiding*

PROBLEM CASE OF EXOPHTHALMOS

DR. ANDREW S. WONG, resident in ophthalmology, presented M. C., a 40-year-old single white woman, with thyroid disease of 21 years' duration, including striking exophthalmos.

In 1931 and 1932, the patient had two subtotal thyroidectomies for thyrotoxicosis. In 1948, the patient fell and fractured her nose, following which, her symptoms increased with her eyes becoming very prominent. She was seen at the Lahey Clinic where she was found to have bilateral exophthalmos, grade III, with loss of upward gaze, diplopia, increased retro-orbital resistance, and injection of the conjunctivas, although she still had good closure of both lids.

A diagnosis of "malignant exophthalmos" secondary to thyroid disease was made. In view of failing vision and steady protrusion of her eyes, an orbital decompression operation was recommended but refused.

The patient was next seen at the Peter Bent Brigham Hospital where she received three courses of radioactive-iodine therapy. A course of ACTH was given between her second and third radioactive iodine treatments by Dr. George Thorn. Following these, the thyroid enlargement disappeared. Because of symptoms of thyroid deficiency which developed, she was placed on thyroid and Lugol's iodine solution.

Two years ago, the patient was seen at the New Haven Hospital where she has been under the care of Dr. John Peters up to the present time. Because of extraordinary distress of her sweating and swollen legs, a bilateral lumbar sympathectomy was performed with relief. Following a viral pneumonia, there was a general return of many old symptoms including exophthalmos and, in addition, a frequency of urination with increasing thirst and obligatory nocturia. The latter was controlled by pituitary and antidiuretic hormone.

The condition of her eyes has since been unchanged. Vision is: O.D., 20/25; O.S., 20/20, corrected—with occasional diplopia and blurring. Exophthalmometer measurements were: O.D., 26 mm.; O.S., 25 mm. (baseline—92 mm.). There was widening of the palpebral fissures, O.D. being greater than O.S. Eye movements showed a general weakness, especially in convergence. Winking was decreased. There were full central and peripheral fields and the fundi were normal.

Parahydroxypropriophenone (CH 47) 150 mg., twice daily, had been administered in an effort to modify the exophthalmos, with no demonstrable effect. Also, the patient was still taking thyroid, Lugol's iodine, digitalis and posterior pituitary powder.

Blood chemistry, in general, remained normal.

Discussion. Dr. John P. Peters, John Slade Ely professor of medicine: The problem of thyrotropic and thyrotoxic exophthalmos is difficult. There is no adequate method to measure thyrotropic hormone.

The exophthalmos long preceded the time mentioned by the patient. This was a long-standing exophthalmos that was fairly well controlled until she had what amounted to a total thyroidectomy with the radio-active iodine. The patient felt that her exophthalmos was improved with the radioactive iodine. Actually the exophthalmos increased. It had not been improved except subjectively. If thyroid had not been given, the exophthalmos would have progressed very rapidly.

The radioactive-iodine dose was excessive and in my opinion not justifiable.

The ACTH in this case seemed to throw the patient into a heart block and a coronary, in any case the coronary came after the ACTH. She had a localized edema of the extremities which improved following a sympathectomy and not thyroid.

Dr. William German, professor of neurosurgery: The important finding in exophthalmos requiring neurosurgical decompression is loss of vision. I have followed the cases of marked exophthalmos in this area and have never seen a case here that required orbital decompression. (Dr. German then outlined on the skull and on photos the techniques in orbital decompression.)

Dr. R. M. Fasanella: While under Dr. Peters' care this patient's exophthalmometer readings have been constant. Her vision has not fallen, there has been no corneal damage, no papilledema, no field loss. There has been no indication for a levator recession, angle tarsorrhaphy or treatment of muscle disturbance, although Dr. David Freeman made a helpful suggestion that prisms might help her reading difficulties.

Dr. C. H. Chang, therapeutic radiologist of the New Haven Hospital: Radiation therapy has been tried in progressive exophthalmos following thyroidectomy since 1929 and, in a few reports, good effects resulted in some cases.

The rationale for orbital X-ray therapy is based on two pathologic features found in biopsy and autopsy materials: (1) Gross enlargement of the orbit muscles, which were the seat of fibrosis, edema and degeneration; (2) round cell infiltration, predominantly lymphocytic, most marked in association with edema and greatest around the vessels.

The lymphocytes are known as highly radiosensitive. The apparent beneficial results of radiation therapy on the inflammatory process are fairly well recognized and the beneficial effect of roentgen therapy in destroying newly formed fibrous tissue is well established in the treatment of recent keloids.

Dr. Merrill C. Sosman of Boston has had some success with irradiation of the retrobulbar portion of the orbit in "malignant progressive exophthalmos," about two or three cases being markedly benefited (personal communication). He uses daily doses through the temporal area, giving 200 or 250 r per day to one side only, alternating the sides until 1,000 r (air) have been delivered. His technical factors are a 250-K.V. constant potential machine with 50-cm. target skin distance and a heavy Thoreus filter.

Recently Arthur Jones of London reported orbital irradiation in 29 cases of exophthalmos, two of which had "malignant progressive exophthalmos," 20 had chronic progressive exophthalmos, and the remaining seven, thyrotoxic exophthalmos.

The best results were found in the cases of "malignant exophthalmos," and clinical improvement of varying degree has been noted in the majority of cases of chronic progressive exophthalmos. Poor results were found in thyrotoxic exophthalmos.

He used the anterior orbital port in addition to the lateral temporal port. A small lead shield was placed over the position of the cornea.

We treated another woman, aged 39 years, with a clinical diagnosis of unilateral chronic progressive exophthalmos following a thyroidectomy for toxic diffuse goiter in November, 1941. We used the Sosman and Jones' technique. Totally, 800 r (air) were given to the lateral orbital region and 200 r (air) to the anterior orbital region, with a lead shield over the cornea, in two courses.

Clinically the patient felt some relief of the pressure effect; however, there has been no improvement in exophthalmos by measurements obtained by Dr. C. C. Clarke.

Dr. Peters: How did they differentiate progressive exophthalmos from thyrotoxic exophthalmos by laboratory methods?

Dr. Chang: In cases of progressive exophthalmos, lower thyroid gland uptake of radioactive iodine, normal protein-bound blood iodine level, and a positive urinary bioassay

for thyrotropic hormone are found.

Dr. I. K. de Suto-Nagy: Were any cataracts observed in those cases treated? I heard that 2.0 r will produce corneal opacity in the laboratory animal.

Dr. Chang: Yes, in Jones' series of 29 cases, one patient (aged 66 years) had been found to have cataracts. Irradiation was by lateral fields only, to a total dose of 1,000 r (air) per field in 25 days. Slight central lenticular cataracts were noted 18 months following the irradiation but it was not known whether these were previously present.

I think Dr. Sosman has made a fair statement: "We try to protect the lens of the eye by directing the radiation just posterior to it, but in most cases it is a question of saving the eye, and you take a chance on getting the lens opacities rather than losing the eyes."

In regard to the experiment that 2.0 r will produce a corneal opacity; personally, I don't know, but I doubt it.

Dr. Fasanella: Dr. Peters, when should radioactive iodine be used in thyroid disorder?

Dr. Peters: I feel that radioactive iodine should be used in carcinoma cases with metastasis if the tumor responds to radioactive therapy. Most other cases should be treated medically.

Dr. C. C. Clarke: Angular tarsorrhaphy might serve to give a better cosmetic appearance and more protection in this case and others.

Dr. Arthur M. Yudkin (in closing): My experience in two cases treated with tarsorrhaphy has not been successful. In both cases the patient begged to have the tarsorrhaphy opened.

October 31, 1952

RHEUMATISM AND THE EYE

DR. JOSEPH IGERSHEIMER, Boston, the guest speaker, presented this subject and illustrated his lecture with many colored slides. An abstract of Dr. Igersheimer's paper follows:

A new era in the field of rheumatic diseases started with anatomic studies of Klinge and Roessle who came to the conclusion that rheumatic diseases are due to characteristic histologic changes in the connective tissue (myxomatous swelling of the ground substance and then fibrinoid alteration or degeneration). A granuloma was not only found in the heart muscle, but in many other organs.

Klemperer and his group found a generalized alteration of the connective tissue in rheumatic diseases, as well as in periarteritis nodosa, acute lupus erythematosus, scleroderma, dermatomyositis, and it was they who coined the term "collagen diseases." It is especially interesting that all these diseases which have so different clinical aspects react especially well to cortisone and ACTH.

Ophthalmology is able to contribute something to the question of how close these different diseases belong together and what separates them. In the question whether rheumatic fever and the chronic forms of arthritis are basically of the same nature, it is noteworthy that such ocular symptoms as iritis or other forms of uveal diseases are extremely rare in rheumatic fever while they are the main ocular localizations in chronic forms of arthritis.

As far as the arthritis and the other collagen diseases are concerned, it is remarkable that in the arthritic group the anterior part of the globe is involved, while in other collagen diseases it is the posterior part of the globe.

The disease which has the highest percentage of ocular pathologic findings is the spondylarthritis ankylopoetica (about 50 percent). Franceschetti is so impressed by this clinical experience that he suggests one should examine the sacro-iliac joints by X-rays in every case of iritis with increased sedimentation rate.

In other types of rheumatoid arthritis an ocular involvement is present in about five

percent of the cases. In chronic arthritic lesions in children (Still's disease), the ocular complication, in the form of an iridocyclitis, often has the specialty of developing a band-like keratopathy with *good* vision.

The clinical manifestations in rheumatic eye diseases are especially characteristic on the sclera. This is especially so of the disease which is called scleromalacia perforans and is almost regularly combined with a chronic arthritis. Also, in more or less deep-seated scleritis or sclerotenonitis, the combination with arthritis is often very striking.

Iritis is the lesion which is most often believed to be caused by a rheumatic disease. The superficial form, a tendency for recurrences, and an absence or a lack of nodules, are generally believed especially characteristic.

It is strange, however, why granulomas should not develop in the iris, whereas, nodular formations are one of the most characteristic signs of rheumatism.

The pathogenesis of the rheumatic diseases in general and the ocular pathology in these conditions are still under discussion. Experimental studies seem to be in favor of a hypersensitivity against different agents, living or dead. The whole question, however, is still hypothetical.

Interesting is the relationship of tuberculosis to rheumatic diseases, especially to those of the ocular apparatus. Poncet has described a tuberculous polyarthritis and there are cases which show arthritic and also ocular lesions which are most likely of tuberculous origin.

Rheumatoid arthritis, however, or other collagen diseases have no tuberculous background, and therefore it is unlikely that the different lesions in the eye in such cases have anything to do with tuberculosis.

Many problems are not solved yet. Not debatable, however, seems the relationship of rheumatic or collagen diseases to the adrenal cortex. The different stages of the cortisone effect upon rheumatic nodules could histologically be demonstrated on the sclera. It is noteworthy that an acute, even a severe iritis, is in most cases well and quickly influenced by cortisone, while a granulomatous uveitis often does not show any real improvement.

COLLEGE OF PHYSICIANS OF PHILADELPHIA

Section on Ophthalmology

March 20, 1952

Dr. George F. J. Kelly, *chairman*

Exophthalmos associated with disturbances in pituitary-thyroid axis

Experimental considerations

Dr. William McK. Jefferies, Western Reserve University School of Medicine, Cleveland, Ohio, reviewed the problems involved in the study of exophthalmos in experimental animals and gave a résumé of the findings derived from this type of investigation up to the present time.

After a brief discussion of the various agents which have been used to produce exophthalmos in animals and the reasons why guinea pigs have proven to be the most suitable subjects for this type of experimentation, the evolution of an accurate instrument to measure intercorneal distance in animals and a reliable technique for producing exophthalmos in guinea pigs was recounted.

Studies of the mechanism of production of exophthalmos by injections of pituitary extract were reported. Several investigators have found that an increase in the content of fluid, fat, and connective tissue of the retro-ocular tissues is involved and that the fraction of pituitary extract containing thyrotropic hormone shows the greatest exophthalmic activity. Nevertheless, by the use of pituitary extract in which the thyrotropic factor has been almost, if not completely, inactivated by iodination, it has been shown that exophthalmic effects in guinea pigs cannot be attributed to activity of the thyro-

tropic hormone alone. Further studies of a similar nature have indicated that the production of exophthalmos is not directly related to the fat-mobilizing effects of pituitary extract.

Other experiments have exonerated the adrenocorticotropic hormone and the follicle-stimulating hormone of any direct relationship. Hence, although the exophthalmic factor exists in close association with the thyrotropic hormone in pituitary extract, it is not this hormone, and its true nature still remains obscure.

Clinical problems

Dr. Edward Rose spoke on the clinical problem. The ocular changes of Graves' disease were briefly described and mention was made of their variability and the gradations of their severity. Lack of knowledge concerning the pathogenesis of these changes is indicated by the numerous theories which have been advanced. Dr. Rose reviewed the more important of these.

He analyzed clinical experience with a number of cases followed for long periods of time, and from this experience drew certain inferences regarding the classification of the syndrome.

This experience casts some doubt upon the acceptability of the separation of exophthalmos into thyrotropic and thyrotoxic types. The frequency of allergic reactions in patients with this syndrome poses the possibility of local tissue hypersensitivity of the orbital contents as a factor in its pathogenesis.

Endocrine and other nonsurgical methods of treatment were reviewed and the difficulty of evaluating such therapeutic measures was emphasized.

Neurosurgical problems

Dr. Rudolph Jaeger described the neurosurgical problems. He said that, as a point of practical information, it is of interest that the neurosurgeon sees no cases of exophthalmos in association with gross surgical disease of the hypophyseal area. Adenomas of the pituitary, destructive disease of this body from cancer or abscess, Rathke duct tumors, and tumors of the hypothalamic region cause no proptosis. Only invading meningiomas or malignant tumors of the retro-orbital bone cause forward protrusion of the eyeball by local congestion and mass increase of the periorbital and intraorbital structures.

The neurosurgeon's interest is concerned with (1) preservation of vision through the prevention of corneal ulcerations and opacities; and (2) the improvement of the appearance of the patient.

The first may be effected by suturing the eyelids together for almost the full length of the lids after denuding the lid edges, being careful not be disturb the lacrimal puncta. Removal of the sutures after 10 days will leave the lids tightly grown together. It assures the optimum condition for corneal healing. Since spontaneous recovery is often possible, when the patient is otherwise inoperable, this maneuver may preserve vision until time effects the cure.

The second neurosurgical maneuver for the treatment of this deformity is intracranial decompression of the orbit by removing its roof (Naffziger operation). In severe cases this can easily be combined with the removal of the lateral orbital wall (Krönlein operation), thereby assuring the maximum decompression possible. When the proptosis is severe and shows no tendency to subside, this operation should be performed. It carries only a slight risk and the cosmetic result is good. Both sides may be operated on at the same time.

Slides were shown of the following: (1) Technique for eyelid closure; (2) patient with malignant exophthalmos before and six and nine months after closing of lids. Spontaneous cure occurred. The patient was elderly and had high blood pressure and diabetes. The technique of intracranial decompression and views of the patient before and after operation were shown by motion pictures.

OPHTHALMIC PROBLEMS

DR. EDMUND B. SPAETH reviewed the ophthalmologic problem.

From the standpoint of surgical therapeutics, there is a definite necessity to differentiate between the basic etiologic factors; by this is meant the consideration of a thyrotoxic state, a thyrotropic basis, or, possibly, a situation in which both are factors.

The eye signs and symptoms of the thyrotropic state can be called quite properly pituitary exophthalmos. In those cases having characteristics of both, the thyroid dysfunction, if that is present, fails to depress pituitary secretion and, as Herman said, "Obviously these cases will present the greatest therapeutic problems." Each of these three types has different surgical indications. These types are not related, wholly, to degrees of severity in the basic disease. They are essentially different diseases.

The fundamental orbital pathologic processes in exophthalmos is significant.

In thyrotropic exophthalmos, the progressive involvement of the eye is entirely disassociated from a thyrotoxicosis. Due to the upset balance of the hormones, water-storage disturbances occur. Because of the confined nature of the orbital space this may have particularly disastrous consequences in that the resulting exophthalmos may progress inexorably.

Thyrotropic exophthalmos has an uneasy proptosis. Phenomena of pain, discomfort, lacrimation, photophobia, and diplopia frequently occur. Patients with this type of exophthalmos often consult an ophthalmologist first. One may find periocular edema, chemosis, and injection of the conjunctivas.

Absence of lid spasm permits free eversion of the lids, and the degree of proptosis is more real than apparent. Often a thick ridge of edematous tissue projects, like a finger, towards the inner canthus. Actually, in thyrotropic exophthalmos, the eyes, not the basic disease, seem to present the major therapeutic problem.

Thyrotropic exophthalmos is always a serious condition—not only difficult to diagnose, but equally difficult to treat. A recent case, as an illustration, is now quiescent, but the patient is in a lamentable condition. The right eye has been enucleated following absolute glaucoma because of massive X-ray therapy for a misdiagnosed retrobulbar neoplasm; the left eye is now immobile due to muscle degeneration, secondary glaucoma (stabilized with miotics) is present, and the lids are in permanent intermarginal tarsorrhaphy.

Before considering local surgical measures—that is, local surgery whose sole purpose is to save the eye—other available therapeutic procedures should be briefly discussed. These are: (1) The administration of thyroid gland or extract to decrease the output of pituitary thyrotropic hormone; (2) to decrease the output of this hormone by modifying the metabolism of the hormone-producing cells, as by X-ray therapy.

Neither procedure is an ophthalmologist's problem; both are procedures to be used alone or as a part of the total treatment of any one case. For example, a simple tarsorrhaphy will prevent damage to the cornea in a severely proptosed eye, regardless of the type of exophthalmos, but neither tarsorrhaphy nor orbital decompression may be sufficient to save the globe in a case of steadily developing intraorbital pressure.

These cases can become so severe, even before the globe is endangered from corneal perforation, as to cause marked permanent degeneration of the orbital muscles, swelling of the optic nerve, and retinal detachment. Long-standing edema can destroy ultimately all orbital muscle tissue, terminating in degeneration and in ocular immotility.

A common finding in these cases, frequently confusing the diagnosis, is the fact that the exophthalmos may be only monocular early in the course of the disease. This can result in the faulty diagnosis of possible unilateral retrobulbar neoplasm or pseudotumor.

The technique for the surgery of the

oculomotor palsies is well understood. In these instances muscle resections are to be performed upon the paretic muscles. The sequence of surgical procedures is a bit more exacting. The basic muscle degeneration so characteristic of these conditions makes surgery upon the conjugate yoke muscles in the form of recessions quite unwise, in fact rather ineffective.

The stationary exophthalmos of arrested thyrotoxicosis, or of severe stationary, and not malignant, thyrotropic exophthalmos, is perhaps largely cosmetic in its demands. It may, however, be otherwise. The progressive type of malignant exophthalmos has an absolutely essential surgical aspect. It may not only be eye-saving, but even life-saving. Such a condition is often a tragic affair and demands both surgical and medical therapy, which frequently is heroic in extent.

The surgery for lagophthalmos, and this may also be quite applicable to a case of stationary exophthalmos in which surgery is needed for cosmetic reasons, is the classical levator recession of Goldstein. This surgery attacks the condition upon the same anatomic basis as that used for correcting paralytic ptosis. It is ideal for quiescent cases.

The levator is detached from the tarsal plate and recessed posteriorward as it lies above the superior rectus muscle and below the septum orbitale, permitting a lengthening of that portion of the lid anterior to the new detachment of the levator to the orbicularis and fascia. One can easily achieve a recession of any amount up to 10 mm.

The surgery may be performed through the skin surface or through the conjunctival surface, as one wishes. It seems that the surgery, when done through the conjunctival surface, has a bit less postoperative reaction.

Some of the cases of stationary exophthalmos, when unaccompanied by lid spasm, can be treated by a surgical procedure which is somewhat easier and which has a much shorter period of necessary hospitalization.

This is the buried silk suture of Axenfeld. It is a simple procedure and should correct cases with a moderate degree of involvement.

Braided white silk is used and one must be most meticulous as to surgical asepsis. I have had failures with this operation in several instances because of a stitch abscess. That will nullify the entire operation and make necessary the removal of the suture.

Mention must be made also of the angle tarsorrhaphy of Wheeler. It is especially valuable when it is necessary to correct the upper and the lower lid to dissimilar degrees.

The surgery for the thyrotropic form of exophthalmos is essentially that of orbital decompression. In these cases it is not uncommon to find that a combination of the various surgical procedures must be seriously considered; that is, the simultaneous use of a levator recession, for instance, combined with an angle tarsorrhaphy, or a levator recession combined with an orbital decompression, or the decompression of the orbit combined with an angle tarsorrhaphy. Occasion has been found to utilize all three of these possibilities.

Much has been written about the transfrontal decompression. I shall not consider this. The subzygomatic decompression as outlined by Shugrue is a satisfactory procedure. The orbitotomy of Krönlein is not necessary. The decompression which the Shugrue technique furnishes is quite adequate.

In the Shugrue technique one must be certain not to enter the cranial cavity through the sphenoidal bone so as not to injure the maxillary nerve at the lower part of the operative field. The purpose of an orbital decompression, as Herman stated so well, "Is to relieve edema and congestion in the orbit, and thereby to restore adequate drainage of the extraocular tissues as well."

Malignant exophthalmos, as such, can be nicely treated with orbital decompression; no other treatment but orbital decompression is successful in these serious situations. In my experience there is no great difference in the results obtained from the transfrontal ap-

proach and uncapping the roof of the orbit as it lies in the frontal fossa, and the subzygomatic approach and orbital decompression as just described.

Cases have been presented in which a transfrontal orbital decompression has been done on one orbit, and a subzygomatic decompression on the other. There seemed to be no difference in the degree of correction obtained.

To recapitulate from an ophthalmic standpoint, the surgical procedures, which have to do with the ocular manifestation of thyroid dysfunction are essentially four in number. They include: (1) A levator recession, (2) an angle tarsorrhaphy, (3) the treatment of the muscle disturbance and the probable sequence of necessary surgical procedures for this, and (4) an orbital decompression when indicated.

Discussion. Dr. George F. J. Kelly: It is not quite clear to me just what Dr. Jaeger has done. From the picture, it is evident that the skull was opened on the right side, and the right orbital roof was partially removed. The picture further showed improvement in the position of both eyes. I would like to know whether both orbits can be reached from the one side, or did this patient have two separate operations?

Dr. Rudolph Jaeger: Yes, the patient had a second operation. You may do both sides at the same time, but this man had two operations.

Dr. Francis Heed Adler: This is one of the clearest expositions of an extremely difficult subject I have heard or read, but perhaps because of its clarity and honesty, it takes away one of our secure diagnostic props. The work of Mulvaney made us feel confident that we had a means by which we could differentiate cases of so-called thyrotropic exophthalmos from thyrotoxic exophthalmos, and thereby aid the clinician in deciding what cases it was admissible to submit to thyroidectomy, and what cases it was probably wise to withhold from surgery.

Dr. Rose has pointed out that there is not a sharp line of distinction between these two types of cases and that, as far as the basal metabolic rate is concerned, this may be elevated in both, and in fact generally is.

We have been led to suppose that the cases of thyrotropic exophthalmos were characterized by edema of the lids and conjunctiva, rapidly increasing exophthalmos, and frequently an associated paralysis of the ocular muscles. In unilateral cases the paralysis of the affected eye is generally of the muscles of upward gaze.

As ophthalmologists, we have a particular problem in having to separate cases of unilateral, so-called thyrotropic exophthalmos from cases of orbital tumor and also from cases of so-called chronic orbital myositis described by Dunnington, Berke, and others. I have always suspected that this latter disease is in reality due to thyrotropism.

To abolish these two clean-cut entities, even though cases are seen which fall in between the two extremes, is going to make it more difficult for the ophthalmologist to give an opinion as to what cases will progress if thyroidectomy is done. Nevertheless, we have to face facts, and I hope that before long those who have been working on this problem will have something better to substitute for Mulvaney's scheme, which it now appears is not based on too firm a footing.

Dr. Irving H. Leopold: The orbital tonometry of Copper has been suggested as helpful in the differential diagnosis of these conditions. Has any member of the panel had personal experience with this diagnostic tool?

Is there a difference in incidence of malignant exophthalmos following the use of propyl-thiouracil, radioactive iodine, or thyroidectomy for control of overactive thyroid signs?

Dr. Alfred Cowan: We know that in many instances, persons with exophthalmos become blind from glaucoma. Of course, it is a secondary type of glaucoma. In spite of this we never hesitate to use cycloplegics in persons with protosis or exophthalmos.

Maybe someone can explain why glaucoma is not produced in these cases.

Dr. William McK. Jeffries: I appreciate the referral of these clinical questions to me, since our experience with exophthalmos has not been confined entirely to guinea pigs.

We also have encountered very unpredictable effects in patients. I have seen three cases treated with radioactive iodine. One of these was that of a man, aged approximately 40 years, with proptosis, chemosis, ophthalmoplegia, and moderate hyperthyroidism with a basal metabolism rate of plus 40—a classical picture of "malignant exophthalmos."

After receiving radioactive iodine therapy, he failed to return to the clinic until about six months later, at which time he had the classical features of myxedema, yet his eyes had not only not become worse, but were actually greatly improved.

It would seem, therefore, the occurrence of myxedema in these patients is not invariably detrimental to their ocular status, further suggesting that the behavior of the thyrotropic hormone is not the whole story. Nevertheless, there has been a sufficiently large number of patients with ophthalmopathic Graves' disease who have experienced aggravation of their exophthalmos upon becoming hypothyroid to indicate that it is best to avoid this development.

The other two patients who received radioactive iodine therapy in our clinic experienced improvement of their ophthalmopathy, but I have heard of one case so treated elsewhere in which the eyes subsequently became worse.

Subtotal thyroidectomy seems to be the type of therapy most likely to be followed by worsening of the eyes, so it is generally considered to be contraindicated in these cases. The administration of antithyroid drugs such as propyl thiouracil, which enhance stimulation, theoretically would not be good for the eyes, and there have been reports of aggravation of ophthalmopathy while patients were receiving them. Hence we agree with Dr. Rose in preferring to use a combination of potassium iodide and thyroid in treating these patients.

The reports that practically every patient experiences an increase in prominence of the eyes after subtotal thyroidectomy might be explained by a concomitant gain in body weight. Unfortunately, these changes in eye measurements in humans have not been correlated with the body weight. It seems possible that the slight increase in prominence of the eyes under such circumstances might be a manifestation of better general nutrition rather than a result of increased pituitary stimulation. None of the cases so followed in these reports actually developed severe ophthalmopathy.

We tried cortisone in a dosage of 100 mg. daily for two weeks in one patient with ophthalmopathic Graves' disease without appreciable effect. Other workers have encountered similar disappointments with ACTH and cortisone after the initial encouraging reports, so these two hormones do not appear to be significant additions to the therapy of this very puzzling clinical entity.

Dr. Edmund B. Spaeth: To answer Dr. Cowan's question relative to "the reason for the onset of secondary glaucoma, in these conditions."

I would like to defer to Dr. Mann in her analysis of this ocular pathologic process in exophthalmos. I think her rationalization is quite reasonable. She likened it to the secondary glaucoma from orbital conditions in venous disturbances, the fibroses, in pulsating exophthalmos, and in pseudotumor. This is not secondary glaucoma resulting from a perforation of the globe. In those situations anything can occur. Dr. Mann's thought was that it was due to closure—slow, inexorable, and progressive—of the major venous channels in the orbit as represented by the vortex veins. This is the only answer I have.

M. Luther Kauffman,
Clerk.

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THE ELECTRONIC TONOMETER

In preparing to study the rate of outflow of aqueous humor by the method of tonography, it was noted that the position of the pointer on the current model of the Mueller electronic tonometer was subject to changes apparently independent of the intraocular pressure being examined.

It is known that electronic devices may sometimes be highly sensitive to voltage fluctuations in their power supply. Furthermore, line voltages may vary considerably in hospitals or laboratories where X-ray apparatus, elevator motors, and other heavy equipment are in use.

Inasmuch as the results of any quantitative research can, in general, be no more accurate than the individual data involved, we considered it a necessary preliminary to determine to what degree electronic tonometer readings change with the voltage supply.

A variable voltage transformer was used in order to be able to set the voltage to the electronic tonometer at will. With an initial power supply of 115 volts, the instrument was calibrated in the usual manner. The

footplate assembly was then clamped rigidly in place, and the level of the plunger fixed by means of a micrometer screw. Finally the voltage was altered. It was found that the tonometer reading could vary considerably with relatively small changes in voltage.

As an example, the plunger was set to give a tonometer reading of 3.0 after calibration at 115 volts. Then the voltage was changed, and the following readings were obtained:

Voltage	Electronic Tonometer Reading	Intraocular Pressure (mm. Hg)
95	1.6	34.1
100	1.8	33.6
105	2.1	31.5
107.5	2.3	30.6
110	2.5	29.6
112.5	2.8	28.1
115	3.0	27.1
117.5	3.2	26.4
120	3.4	25.7
125	3.7	24.6

The intraocular pressures, assuming a 5.5-gm. weight, were obtained by interpolation from the chart* supplied with the instrument. It will be noted that a change of five volts in the power supply, a common occurrence with ordinary wiring, can simulate a difference of several millimeters of mercury in the deduced intraocular pressure. This effect was not strictly consistent from day to day, and even greater discrepancies have been observed.

Apparently the potential differences between the ground wire and the current supply wires may also be important factors, and capacitance changes seem to have a great bearing on the readings. In any case, it seemed important to eliminate or at least to minimize ordinary fluctuations in line voltage.

This was satisfactorily accomplished by application of a constant-voltage transformer. Thus a power supply varying between 90 and 130 volts was readily stabilized to within 0.5 percent of 115 volts.

These remarks are not intended to detract from the desired advantages of the electronic tonometer, for example, the large scale. However, I do wish to emphasize that a constant voltage supply and careful attention to other electrical details are essential for accurate results. This is especially important in tonography, where recalibration during the recording could completely invalidate the results obtained.

S. I. Askovitz.†

* Chart for electronic and Schiøtz tonometers, approved by the Committee on Standardization of Tonometers of the American Academy of Ophthalmology and Otolaryngology, January, 1948.

† From the Ophthalmology Research Laboratory, Albert Einstein Medical Center, Northern Division, Philadelphia.

THE PAN-AMERICAN INTERIM CRUISE

Approximately 170 ophthalmologists, accompanied by a heterogeneous assortment of wives, children, friends, and an occasional mother-in-law, boarded the *M. S. Italia* at New Orleans on January 31st for a two-week ophthalmological odyssey in the Caribbean Sea.

Despite the competition of various shipboard delights, the scientific program was surprisingly well attended, due in part to the excellent and unique character of the program planned under the direction of Dr. James H. Allen.

At nine o'clock each morning when the ship was not in port, members had a choice of an informal demonstration of slitlamp microscopy by Dr. Daniel M. Rolett or a discussion of practical optics by Mr. Milton Spew. From 10 o'clock to 12 noon, various panels were held. One of the most popular was a four-session panel on cataract under the chairmanship of Dr. Daniel B. Kirby.

Other panels were conducted on diseases of the lids, medical ophthalmology, intraocular foreign bodies, extraocular muscles, glaucoma, and refraction, with Dr. E. R. Veirs, Dr. W. A. Mann, Dr. Norman L. Cutler, Dr. R. N. Berke, Dr. E. W. Griffey, and Dr. James E. Lebensohn serving as chairmen of these respective panels.

It was the hope of Dr. Moacyr E. Alvaro, president of the Pan-American Association

of Ophthalmology, which sponsored the cruise, that a new type of meeting might be evolved, and this hope was realized. A successful formula has apparently been developed for this type of meeting.

Formal presentation of four or five excellent related talks during the first hour was followed, during the second hour, by discussion, by a panel of arbitrarily selected members, so that, during the meeting, each member attending had an active, planned participation in at least one session in addition to the stimulating effect of general informal discussion. The caliber of the discussions was excellent and all members felt that the scientific aspects of the meeting were exceedingly successful.

Technical exhibits were of necessity limited but representatives of a firm of instrument makers and a dealer in medical books were on hand with extensive displays.

Pleasant luncheons and meetings with local ophthalmologists of Kingston, Caracas, San Juan, and Havana, including in the latter port a reception at the Havana Country Club, given by the Cuban Minister of Health, followed by ceremonies at the Academy of Science, gave opportunity to meet professionally and socially many of our Latin American colleagues. The placing of a wreath at the tomb of Bolivar, liberator of South America, and the unveiling of plaques at other centers were gestures helpful to the cementing of Pan-American ophthalmological relations.

It is to be regretted that the secretary general, Dr. Thomas D. Allen, who had conceived and organized the cruise, was unable to make the trip because of ill health. His substitute, Dr. Daniel Snydacker, was helpful in keeping the meeting and members under control.

The calm blue waters of the Caribbean were conducive to hours of relaxation in the warm sun, and the social life on board ship and off was by no means neglected. All participants felt that this more or less experimental type of meeting had been an enormous success and that it would be the forerunner of many future such Pan-American interim meetings.

William A. Mann.

ARNOLD LOEWENSTEIN

(A Note of Appreciation)

The recent passing of Arnold Loewenstein in Glasgow, Scotland, will bring back memories to a group of U. S. Army ophthalmologists, who, as part of a Post-Hostilities Course in Ophthalmology, began on V-J Day, 1945, a week of lectures and demonstrations at the Tennant Institute of Ophthalmology in Glasgow. This course of instruction, under the supervision of Professor Riddell and Professor Ballantyne, contained several lecture demonstrations by Dr. Loewenstein.

As visitors and students we were impressed by his sparkling wit and good humor, his tremendous enthusiasm for teaching and investigation, and his vast reservoir of ophthalmic knowledge.

A review of my notes made at the time shows that Dr. Loewenstein's ideas on his favorite subjects—allergy, tuberculosis, sympathetic ophthalmia, the phakomatoses, and others—were based on his own vast clinical and laboratory experience.

A sense of gratitude for the inspiration received from Dr. Loewenstein as well as from the other lecturers in this brief course prompts this expression of regret at his passing.

F. Phinizy Calhoun, Jr.

XVII INTERNATIONAL CONGRESS OF OPHTHALMOLOGY

September 13 through September 17, 1954
New York City
Waldorf-Astoria Hotel
Subjects for discussion: *Glaucoma* and *Uveitis*

Dr. Arnold Loewenstein (with hand in coat), Professor Riddell, and Professor Ballantyne with a group of U. S. Army ophthalmologists in Glasgow, Scotland, in August 1945.

CORRESPONDENCE

REQUESTS FOR REPRINTS CONCERNING STRESS AND THE ADAPTIVE HORMONES

Editor,

American Journal of Ophthalmology:

In perusing the current literature with which this JOURNAL is concerned, we note that an ever-increasing number of its articles deals with problems pertaining to research on "stress" and the so-called "adaptive hormones" (ACTH, STH, corticoids, adrenergic substances, and so forth).

We are writing you because, in our opinion, the success of research in this complex and rapidly developing field largely depends upon the prompt availability and evaluation of relevant publications, a task for which we should like to solicit the assistance of your readers.

In 1950, our Institute initiated the publication of a series of reference volumes entitled *Annual Reports on Stress* (Acta Medical Publishers, Montreal) in which the entire current world literature is surveyed every year (usually between 2,000 and 4,000 publications). Up to now, we had to compile the pertinent literature partly from medical periodicals, monographs, abstract journals, and partly from reprints sent to us by the authors themselves.

Of all these, reprints proved to be the best source of data which we felt deserved prompt attention in our annual reports. Hence, in the past, we have sent out several thousand individual reprint requests to authors of whom we knew that they are currently engaged in research on stress and allied topics. Even this procedure did not give us the wide coverage which would be

desirable, because it is materially impossible to contact all these authors individually and it often takes too much time to get the requested reprints.

It is evident that, in order to insure prompt inclusion of publications in the annual reports, these surveys must develop into a coöperative effort between the authors of original papers and the reviewers. This coöperation was greatly enhanced of late by the publication of announcements, in several medical journals, encouraging investigators interested in stress research to send us their reprints for this purpose as soon as they become available.

We should be grateful if, by the publication of this note, you would also bring this problem to the attention of your readers.

(Signed) Hans Selye,
and
Alexander Horava,
Institute of Experimental
Medicine and Surgery,
University of Montreal,
Montreal, Quebec.

Alleged effects of tinted lenses to aid vision in night driving by reducing ultraviolet light

Editor,
American Journal of Ophthalmology:

Recent advertisements state that ophthalmic lenses excluding ultraviolet light aid vision by hastening dark adaptation in night driving. Reasons given are of a technical nature unfamiliar to us. Will the statement stand criticism, or is it another exaggeration? The advertisements are open to criticism.

First, they do not specifically state, but imply, that the tinted lenses may be worn both day and night. Even if ultraviolet light during the daytime did prevent rapid dark adaptation (which it does not), vision at night would be better through clear glass than it would through the tinted lenses. Any tinted lens of transmittance of 80 percent or less is dangerous for use in night driving.

Second, the advertisements depend on the hypothesis that ultraviolet light slows the dark-adaptation rate for several hours after exposure. It assumes that, if the individual is protected against ultraviolet from the sun or from fluorescent lights, the dark adaptation that night will be rapid. When during night driving, the eyes are light adapted by the light from oncoming headlights, dark adaptation recurs quickly after the car passes, if ultraviolet light has been avoided.

It has been amply proven that excess visible light during the day reduces the speed of dark adaptation that night,[1, 2] but even this cannot be prevented by a tinted lens. Extremely dark glasses of 10- or 12-percent transmittance are required. However, dark adaptation is not similarly affected by any invisible light.[3] Not only is light in the ultraviolet part of the spectrum below 410 mu incapable of stimulating the retina but, in presence of cornea and lens, such light cannot even reach the retina.[4, 5] If the tinted glass advertised cuts out ultraviolet, which is already cut out by normal cornea and lens, how can it possibly affect the speed of dark adaptation?

Third, the advertising depends on the supposition that driving a car at night involves chiefly scotopic or dark-adapted vision. It certainly does not.

Cone dark adaptation begins at twilight, 1.0 to 0.1 mL (millilamberts, 1.0 mL = 0.929 foot-candles), and requires only a few seconds time. Rod dark adaptation begins at about 0.1 mL, which is deep twilight. At the illumination levels reflected from objects on the road under automobile headlight illumination, dark adaptation does not proceed beyond one-tenth its full extent. The normal automobile illumination is about 0.1 mL at a distance of 280 feet in front of the car. Illumination of 0.01 mL, equal to full moonlight, is produced by automobile headlights 900 feet ahead, and even that would produce

only half dark adaptation.

Therefore, the advertisements which state that tinted lenses which reduce ultraviolet light are helpful for night driving are misleading and exaggerated.

(Signed) Paul W. Miles,
Saint Louis, Missouri.

REFERENCES

1. Peckham, R. H.: The protection and maintenance of night vision for military personnel. Am. J. Ophth., **30**:1588, 1947.
2. Peckham, R. H., and Harley, R. D.: The effect of sunglasses in protecting retinal sensitivity. Am. J. Ophth., **34**:1499, 1951.
3. Wald, G.: Alleged effects of near ultraviolet on human vision. J. Optic. Soc. America, **42**:171, 1952.
4. Wald, G.: Science, **101**:653, 1945; Doc. Ophth., **3**:94, 1949.
5. Ludvigh, E., and Kinsey, V. E.: Science, **104**:246, 1946; Kinsey, V. E.: Spectral transmission of the eye to ultraviolet radiations. Arch. Ophth., **39**:508, 1948.

BOOK REVIEWS

LE MANIFESTAZIONI OCULARI DELLE MALATTIE DA VIRUS E DA RICKETTSIE. (The Ocular Manifestations of Viral and Rickettsial Diseases.) By V. Cavara and G. B. Bietti. Bologna, Licinio Cappelli, publisher, 1952. 834 pages with index, references, and numerous illustrations, tables, and charts. Price: Not listed.

This extensive monograph was presented to the 39th Congress of the Italian Ophthalmological Society at Turin on October 8, 1952, and is the only complete review of the subject in the literature. The authors reviewed the now very extensive ophthalmologic literature dealing with viruses and rickettsiae, and covered the essential points of the still more extensive microbiologic literature. The completeness of the references, for each of which the subject is given in the original language of the report and the author's initials are included with his name, greatly enhances their value to the student.

In an introductory section L. Scalfi discusses the general biologic and morphologic characteristics of these unusually interesting disease-producing agents whose importance in ophthalmology has been increasing steadily.

The first chapter, written by Cavara, deals exhaustively with the ocular manifestations of herpes-simplex virus and contains 46 excellent illustrations, including two in color.

The next most important chapter, written by Bietti, is on trachoma. It is 158 pages in length and has six illustrations, including one large color plate; the latest data on antibiotic therapy is discussed.

A third chapter of special interest, written by Cavara, deals with the ocular malformations resulting from maternal infection with viruses during the period of gestation.

Other chapters are concerned with such viral diseases as herpes zoster, molluscum contagiosum, verruca, epidemic keratoconjunctivitis, inclusion conjunctivitis, and Newcastle disease conjunctivitis.

Reflecting their lesser role in ocular pathology the rickettsial diseases occupy only one chapter of the monograph. Among those discussed are typhus fever, tsutsugamushi fever, rickettsial pox, and Q fever.

The last section of the monograph is devoted to ocular diseases which are probably viral in nature but have not yet been proven to be. These include a large number of diseases with major eye involvement, such as ocular pemphigus, Reiter's disease, Behçet's syndrome, erythema multiforme, Harada's disease, the Vogt-Koyanagi syndrome, and sympathetic ophthalmia; and a number of diseases with minor eye involvement, such as Hodgkin's disease, leukemia, and neuromyelitis optica. Concluding the monograph is a table summarizing the ocular tissues involved in the various viral and rickettsial diseases which have been discussed.

It is hoped that an English translation of this valuable work will soon become available.

Phillips Thygeson.

La Permeabilita Corneale. Ricerche storiche—critiche—sperimentali. By Giuseppe Scuderi. Torino, Edizioni Minerva Medica.

Both the author and the publishers are to be congratulated for their work in producing this excellent and beautiful book. The author is well and favorably known for his scholarly work on corneal permeability, but it is too often taken for granted that once a book is written, everything else somehow happens automatically. No nod of appreciation is given for countless plans and decisions that someone must make if the book is to take form.

It seems appropriate, therefore, first to salute the publisher for agreeable typography, perspicuous arrangement, and attractive binding. However, much as the reader is helped by the form of the book, the matter of prime importance is the content which has been given form and for that the author must be praised.

In making this book Professor Scuderi has used the ideas which emerged from his own extensive investigations and the data which other investigators have recorded in the literature and arranged them to form a systematic account of the factors which bring about and modify corneal permeability. The bibliography is conspicuously free from nationalistic bias; to be sure there are many more references to Italian literature than there would be in a similar American study, but the English, French, German, and Japanese languages are generously represented. The excellent illustrations are not numerous, but each illustrates; it does not merely decorate.

In the first chapter the author outlines factors which influence the passage of substances through the cornea and conjunctiva. In the next, the character of absorption and the method of its study are described. Methods may be colorimetric, chemical, physiochemical, or biologic. The experimental contributions to this study of the paths of absorption of collyria are discussed and summarized.

In a fourth chapter the action of various physiologic factors which are concerned with permeability, such as transparence, fluid exchange, nutrition, respiration, and chemical constitution, are evaluated. The characteristics of permeability peculiar to each of three corneal strata—the epithelium, the stroma and the endothelium—have fundamental importance. The epithelium and the endothelium are selective in permeability. The stroma is of no essential importance and the two vitreous membranes are barriers through which fluids pass by simple diffusion.

The experimental work which has contributed to an understanding of this relationship is described and the significance of the various factors is perspicuously summarized. An extensive chapter is devoted to the pharmacology of corneal permeability, and two others to an analysis of chemical, physicochemical, and physical factors.

F. H. Haessler.

Disorders in Perception. By Morris B. Bender, M.D. Springfield, Illinois, Charles C Thomas, 1952. 109 pages, five figures, bibliography, and index. Lexide. Price: $3.00.

The value of simultaneous stimulation, on which this original study is based, was independently rediscovered by Bender during an examination of a patient with a shrapnel injury of the brain sustained some months previously. Standard perimetry revealed no field defect, but targets simultaneously exposed on either side of the fixation point disclosed a homonymous hemianopia.

Though the neurologist, Oppelreuter, used this method in visual field studies in 1917, its importance in diagnosis had been overlooked. Continued investigation showed that double stimulation exposed field defects which only later became evident with the single object test. The gradients in excitability produced

by cerebral lesions may be thus demonstrated in the half, quadrant, or sector of the visual field. Often a rapid to-and-fro movement perceived in the defective area becomes invisible as soon as a new stimulus is introduced in the normal field.

In some cases of homonymous hemianopia, the patient is able to see a small, faintly luminous target in the blind half-field in a completely dark room but not at all under a surrounding illumination of five foot-candles. In this instance, the extinction of sensation results from the simultaneous stimulation of the illuminated background in the remaining normal half-field.

In general, whether extinction or merely obscuration ensues depends on two factors—the intensity of stimulation on the normal side and the threshold in the affected portion. Being a neurologist, the author is primarily concerned with the general aspects of double stimulation, and has found the method applicable also in disturbances of auditory, cutaneous, proprioceptive, and taste sensations. It is of most value in cerebral lesions, of lesser value in affections of the spinal cord, and of no value in peripheral nerve disease.

James E. Lebensohn.

OFFICE MANAGEMENT OF OCULAR DISEASES. By William F. Hughes, Jr., Chicago, The Year Book Publishers, 1953. 452 pages, 52 tables, 120 figures, chapter references, index. Price: $9.00.

In 1947, The Year Book Publishers brought out a useful book for the practicing ophthalmologist, *Office Treatment of the Eye*, by the late Elias Selinger. It met with well-deserved success and the edition was soon exhausted.

After Dr. Selinger's death, the publishers fortunately turned to Dr. Hughes to fill the demand. Dr. Hughes, professor and head of the Department of Ophthalmology, University of Illinois, College of Medicine, is eminently qualified by training and experience to prepare such a manual. This he has done in a most excellent and practical fashion.

The reader is told exactly what to do, and how to do it, in given ocular conditions. This is most helpful, especially to the physician of little experience who is confronted with a problem that is not yielding to his own approach.

It is difficult to single out special chapters for special commendation. However, a unique one on radiation treatment, which is Dr. Hughes' own particular field of investigation, is particularly noteworthy. Another very valuable chapter and one that is certain to be well thumbed is that on "Ophthalmic Formulary," written with the assistance of Dr. Clarence Hans, Jr.

Every ophthalmologist should have a copy of this book at home and also one in his office.

Derrick Vail.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

8

UVEA, SYMPATHETIC DISEASE, AQUEOUS

Danis, Pierre. **Angioma of the choriod.** Arch. d'opht. **12**:487-509, 1952.

The author presents in detail, complete with descriptions of microscopic preparations, two cases of angioma of the choroid, one in which the choroidal angioma was the only lesion, and the other with an associated angioma of the face. The first patient was a woman of 32 years, with retinal detachment, and with the posterior portion of the globe filled with heavily vascularized yellowish masses. The eye was removed because of suspected intraocular tumor. Histologic examination showed a choroidal angioma lying between the papilla and the macula, which herniated into the scleral orifices occupied by a ciliary nerve. The retina was totally disorganized and detached except over the angioma where it was adherent through a fibrinous plaque. There were numerous signs of secondary glaucoma, including anterior synechiae.

The second patient was a woman of 21 years with an extensive facial angioma and severe and progressive neurological signs. Skull plates showed important parieto-occipital calcifications. Ophthalmoscopic examination during life was negative but at autopsy an angioma of the choroid without glaucoma or retinal detachment and meningeal and cerebral angiomata were discovered.

Danis then reviews the literature on choroidal angioma and points out that isolated choroidal angioma usually occurs in association with hydrophthalmos, secondary glaucoma, retinal detachment, or expulsive hemorrhage following cataract extraction. Angioma of the choroid associated with angioma of the face is most often accompanied by hydrophthalmos, primary or complicated with cataract and retinal detachment, by detachment of the retina with glaucoma, and by microphthalmos, in addition to the secondary glaucoma, expulsive hemorrhage, and simple retinal detachment which accompany the isolated type of angioma. In 40 percent of the histologically verified cases reported in the literature there were associated facial lesions. If all cases of chronic simple or secondary glaucoma, retinal detachment, or expulsive hemorrhage were studied histologically, an appreciable

number would be found to be due to isolated choroidal angiomata. (7 figures, 3 tables, 88 references)

Phillips Thygeson.

Pisano, E. **Clinical observations of a case of Doyne's choroiditis.** Boll. d'ocul. **31**:559-565, Sept., 1952.

Doyne's choroiditis was seen in a 51-year-old man four of whose siblings were living and eight had died very early in life. The typical fundus picture is displayed in a colored drawing. The disturbance was symmetrical in both eyes, noninflammatory and paramacular. There was no hemeralopia, visual acuity was 10/10 in each eye, the visual fields were normal, and there was slight mental deterioration. (1 figure, 11 references)

K. W. Ascher.

Walker, Glenn L. **Central serous choroidosis.** J. Iowa M. Soc. **42**:303-305, July, 1952.

The benign, noninflammatory, serous, macular lesion of central serous choroidosis is discussed. The term is descriptive of this condition; the underlying cause is a disturbance of the choroidal circulation in the vascular network for the affected macular areas. This lesion is similar in its origin to senile disciform degeneration of the macula. The diagnostic features of the disease, a differential diagnosis and some brief general statements regarding treatment are presented. (8 references)

James V. Bolger.

Wilkowa, Maria. **Ocular changes in sarcoidosis.** Klinika Oczna **22**:147-152, 1952.

Sarcoidosis in a man 26 years of age is described. It was characterized by the bilateral occurrence of lesions in the anterior portion of the uvea and the parotid glands, and temporary paralysis of the facial nerves. (1 figure, 9 references)

Sylvan Brandon.

9

GLAUCOMA AND OCULAR TENSION

Braley, Alson E. **Medical responsibility to the glaucoma patient.** J. Iowa M. Soc. **42**:519-522, Nov., 1952.

One person in 40 above the age of forty has undiagnosed chronic simple glaucoma. Early diagnosis is essential. All patients with narrow anterior chambers should be given provocative tests. The dark room test is advocated in narrow-angle and the tension curve or tonography test in wide-angle glaucoma. Medication should never be discontinued unless the physician can dogmatically say, "you do not have glaucoma." If surgery is indicated, it should be done immediately.

Irwin E. Gaynon.

Heydenreich, Andreas. **Mydriatics for the treatment of pain in absolute glaucoma.** Klin. Monatsbl. f. Augenh. **121**: 168-174, 1952.

The author found that in 12 out of 23 patients with absolute glaucoma a dilation of the pupil relieved the pain and often decreased the intraocular pressure. The other 11 patients remained uninfluenced by the administration of a mydriatic. None of them got worse. This simple method may make it possible to avoid surgery. (3 graphs, 8 references)

Frederick C. Blodi.

Israel, E. B. **The syndrome of glaucomato-cyclitic crises.** South African M. J. **26**:809-810, Oct. 11, 1952.

The syndrome consists of recurrent attacks of unilateral increased ocular tension in the same eye. The attacks come on at irregular intervals and may last from one to 14 days. The congestion varies from barely perceptible color change to beefy red. Keratic precipitates may be present. There are no posterior synechiae. The disc and fundus are always normal and the provocative tests are

always negative. Surgery does not prevent recurrence of attacks. (3 references)

Irwin E. Gaynon.

Jablonska, Walentyna. **Changes in visual fields in hypertension.** Klinika Oczna **22**:119-126, 1952.

The author discusses the pathogenesis and classification of hypertension. Particular attention is paid to conditions causing ocular symptoms. Visual fields were examined in 38 patients with hypertension, 30 of whom had essential hypertension. There were no changes in the peripheral field in 14 cases, in 13 there was slight peripheral narrowing, in 9 there was narrowing of the field and partial loss of temporal field in one eye, and in 2 there was bitemporal hemianopsia. Localization of pathologic changes causing changes in the visual fields is reviewed. (24 references)

Sylvan Brandon.

Kozakiewicz, Angelina. **Some provocative tests in glaucoma and the intraocular tension in the eye with filtrating scars.** Klinika Oczna **22**:103-118, 1952.

The author describes and discusses the technique and the theory of provocative tests. She noticed that the level of the ocular tension was fairly stable when the filtrating scar was under the upper lid and fluctuating when it was in the palpebral fissure. In the latter case the closing of the eyes for 20 to 40 minutes would raise the intraocular pressure. Raising the upper lid of eyes with the scar under the lid and removing the pressure on the filtrating scar would lower the intraocular pressure. The author investigated the reaction of ocular tension in three different types of cases: known glaucomatous eyes without surgery, glaucomatous eyes on which surgery had been performed, and the fellow eye in such patients. The tests were: 1. closing of eyes in the dark room for 40 to 60 minutes, and 2. staying in the dark room with open eyes for 40 to 60 minutes. Closing the eyes raised the tension in only a few cases if no cocaine was used. The dark-room test raised the tension in 20 to 25 percent of cases. In postoperative tests the rise of tension occurred where the operation was not successful. There was a rise of tension in the fellow eye after monocular surgery in both tests if cocaine was used. The increase was less pronounced if procaine was used. (2 figures, 23 references)

Sylvan Brandon.

Leydhecker, Wolfgang. **The use of provocative tests for analysing functional disturbances in glaucoma.** Klin. Monatsbl. f. Augenh. **121**:174-184, 1952.

Provocative tests can be statistically evaluated to prove their reliability for ruling out glaucoma. As each test examines only one single function, a negative result does not rule out glaucoma. The analytical evaluation of the provocative tests evaluates the functional disturbance which must be present in order to give a positive test. If a specific tests is negative, we can at least assume that one specific function of the eye is not disturbed.

The lability test. Various authors have concluded that the lability test is unreliable because it gives different results in the same eye at various times. The author (partly in collaboration with Thomassen) could show that the result of this test depends on the phase during which the glaucoma is tested. If the glaucoma is in the ascending phase (i.e. the tension is rising during the diurnal curve), then the lability test may be strongly positive. If the glaucoma is in the descending phase, the lability test will be invariably negative. The fact that the increase in pressure occurs within 30 seconds and disappears two minutes after the test speaks against a nervous stimulation of the intraocular vessels or an increased aqueous production. Both would not disappear after so

short a time. An obstruction to outflow on the other hand could not produce a pressure increase so quickly. We must then assume that a hyperemia of the eyes, caused by the cuff around the neck, is the reason for the increase of tension. The author also found that the icewater test does not add anything and advises the cuff test alone. In any case, he found a positive result in only 12 to 15 percent of glaucomatous eyes. But this test can give us an indication whether we are in the ascending or in the descending phase. This may be important for other provocative tests, so we can distinguish between spontaneous and provoked pressure changes.

The water-drinking test. Here the author found that 40 percent of glaucomatous eyes gave a positive result (after one-half or 1 liter of water). There is definitely an osmotic change in the blood, and the aqueous can during that time escape through Schlemm's canal only. The result of this test is independent of the phase (increasing or decreasing tension) of the glaucoma and the positive outcome must be based on an organic impedence to outflow.

The mydriasis test. This will be positive when half of the angle is blocked by the iris and is not dependent on the phase of the glaucoma. It does not indicate whether glaucoma is present or not, but whether an acute attack of glaucoma may be expected or not. It is never positive in eyes with wide angles. (6 tables, 43 references) Frederick C. Blodi.

Lijo Pavia, J. **Cortisone and ocular tension.** Arch. oftal. Buenos Aires **27**:121-123, March, 1952.

A 62-year-old woman had iridocyclitis some 40 years ago and now developed secondary glaucoma. The hypertension and the marked pain were reduced by cyclodiathermy operation with a superior iridectomy. The tension was reduced from 75 mm. Hg (Schiøtz) to 20. In order to clear the corneal opacity 25 mg. of cortisone was given every two hours and in two days the tension rose to 35 mm. Hg. The cortisone was withdrawn for five days and the tension became normal. The cortisone treatment was resumed in smaller doses and in seven days the tension rose again to 50 mm. Hg, to fall again when cortisone was withdrawn. The author found no report of a similar effect of cortisone. Joseph I. Pascal.

Monje, Manfred. **Dark adaption in glaucoma.** Klin. Monatsbl. f. Augenh. **121**:199-204, 1952.

The author could show that increased intraocular pressure does not impair light sense. He tested 29 patients with glaucoma on the Zeiss projection adaptometer. Decreased dark adaption in glaucoma is caused by the constricted field or the narrow pupil. (1 graph, 1 table)
Frederick C. Blodi.

Neubauer, Hellmut. **The nonperforating cyclodiathermy according to Grueter.** Klin. Monatsbl. f. Augenh. **121**:9-15, 1952.

With a 0.75 mm.-electrode, coagulations are set in front of the insertion of the external rectus muscle, but not closer than 3 mm. from the limbus. The applications are made after the conjunctiva has been dissected. This should combine an effect on the ciliary body with a partial obliteration of branches of the long ciliary arteries. At Grueter's clinic in Marburg 120 such operations were performed since 1948. It was used as a primary operation and as a last resort. There was success in 63 percent of the cases. (2 figures, 12 references) Frederick C. Blodi.

10

CRYSTALLINE LENS

Orlansky, George J. **Spontaneous rupture of the lens in hydrocephalic epilepsy:**

report of a case. Arch. Pediat. **69**:247-252, June, 1952.

Spontaneous rupture of the right lens in an 11-months-old hydrocephalic epileptic boy is reported. The case history and autopsy findings are discussed. The rupture is ascribed to mechanical compression of the globe. (5 figures, 6 references)

James V. Bolger.

Schmidt, Rolf. **The nonsurgical treatment of cataracts.** Klin. Monatsbl. f. Augenh. **121**:148-153, 1952.

Putzar (Arztl. Praxis III-21) recently described his successes with a new drug in treating senile cataract. This drug contains the protein of lenses of young animals, ascorbic acid, vitamine B_2, potassium iodide and strychnine. The author tested the drug in 20 cases. In none of them did the lenticular opacities, as examined under the slitlamp, diminish. A few patients experienced an increase in visual acuity probably caused by the effect of strychnine on the retina. (2 tables, 13 references)

Frederick C. Blodi.

Wolfe, Otis D. **Glaucoma and cataract.** J. Iowa M. Soc. **42**:522-524, Nov., 1952.

Narrow-angle glaucoma without congestion which can be controlled by miotics will respond to a cataract extraction. In wide-angle glaucoma cataract extraction must be combined with a filtering operation, done either before or at the time of the cataract extraction. A combined filtering operation should be done if anterior synechiae are present. In cases of narrow-angle glaucoma in the congestive phase, a filtering operation should precede the cataract extraction.

Irwin E. Gaynon.

11

RETINA AND VITREOUS

Andersen, S. Ry. **Atypical falciform retinal fold.** Acta ophth. **30**:325-327, 1952.

The characteristic feature of this malformation is a fold of the inner layer of the optic cup, adhering to the abnormally persistent primary vitreous body, particularly the hyaloid artery and the vascular tunic of the lens. The author reports a case of a 52-year-old woman, with a divergent and amblyopic left eye. An examination of the fundus showed a thick vascular cord extending downward and nasally from the optic disc, and then forward becoming attached to the lens at the site of a posterior polar cataract. This is regarded as an atypical falciform retinal fold. The author also describes a case of retinal detachment with a keel-shaped retinal fold and posterior polar cataract; it is pointed out that in such cases the probability of a persistent primary vitreous should be kept in mind. (1 figure, 5 references)

Ray K. Daily.

Berliner, Milton L. **Lamellar resection of sclera in treatment of retinal detachment.** A.M.A. Arch. Ophth. **48**:596-601, Nov., 1952.

The author's operation consists of the removal of tangential strips of three fourths of the scleral thickness in conjunction with diathermy coagulation. The sclera, after resection, is shortened by buried silk sutures. This procedure is recommended in cases of retinal detachment with a poor preoperative prognosis or in cases of postoperative failure with the usual procedures. (1 figure, 7 references)

George S. Tyner.

Cramer, Federico E. K. **Alterations in the fundus of the eye in a case of tuberculous meningitis in a child treated with streptomycin.** Arch. oftal. Buenos Aires **27**:213-221, May, 1952.

The use of the new antibiotics, especially streptomycin in tuberculous meningitis, has reduced the mortality rate of this disease from about 100 percent to about 60. It is important to recognize

the condition as early as possible and a study of the fundus often makes early diagnosis possible. All the changes in the choroid, optic nerve and papilla which may be seen are described in detail.

Joseph I. Pascal.

Diaz-Dominguez, Diego. **Macular degenerations.** Arch. Soc. oftal. hispano-am. **12**:799-992, Aug., 1952.

This exhaustive monograph on the subject is accompanied by a bibliography, which covers the literature of the last 25 years. Summarizing the salient facts which emerge from this voluminous material, it is apparent that there is a close relation between the various forms of macular degeneration and the form of retinitis pigmentosa. The macular lesions are generally classified according to the primary seat of the lesion. The author suggests that the lesions be grouped according to clinical similarities. From this frame of reference, macular lesions can be divided into two large groups. The first group in which the pathologic process involves tissues ectodermic in origin comprises Doyne's choroiditis, the various forms of heredomacular degeneration, the various forms of retinitis pigmentosa, and juvenile and amaurotic family idiocy. This group has in common a disturbance of the pigment epithelium and neuroepithelium, without any significant abnormality of the mesodermal tissues, primarily or secondarily.

The participation of the mesodermic tissues in the dystrophy characterizes the retinal lesions of the second group. It comprises disciform macular degeneration, angioid streaks, Fuchs's spot in myopia, retinitis circinata, Sorsby's dystrophy and cystoid degeneration. The disturbance of the mesoderm is primary in the lamina vitrea in Junius-Kuhnt disease, in angioid streaks, and in Fuchs's spot. It is secondary in the hemorrhages which accompany these lesions, in retinitis circinata and in the edema which occurs to a lesser or greater degree in all these lesions.

The similarity of the ophthalmoscopic pictures during the course of the evolution of the Junius-Kuhnt disease, angioid streaks and retinitis circinata led some authors (Coppez and Danis) to regard them as one disease, in the same manner as disease of tissues of ectodermic origin were regarded by Leber, and later by Behr and François as the manifestations of one clinical entity. There are cases which represent transitional phases between these two large groups, and combine some of the lesions of each. There are families in which some members have lesions of ectodermal tissues, grouped under tapeto-retinal degenerations, and others have lesions of mesodermal tissues, described by Sorsby as exudative lesions. There are also cases in which each eye of the same patient is affected by lesions belonging to a different group. The author inclines to accept the view that all these lesions, termed abiotrophies, as well as retinitis pigmentosa are manifestations of a precocious senility. The lesions of the lamina vitrea in Kuhnt-Junius disease are regarded as similar to wrinkles of the skin. The loss of pigment in the senile macula is a process similar to the graying of hair and senile loss of skin pigment. For this reason retinal abiotrophies are often associated with other degenerative changes; angioid streaks are often associated with pseudoxanthoma elasticum, and various degenerative changes in the nervous system are frequently associated with tapeto-retinal degenerations. Only in the myopes are the lesions limited to the eye. The myopic eye is an aged eye, and presents all the gradations of degenerative changes both of the exudative and tapeto-retinal type. The common factor to both groups is an abnormal localized senility, which is probably a consequence of hereditary changes in the thalamo-

hypophyseal region. (26 colored plates, 292 references) Ray K. Daily.

Fabian, Gerhard. **Temporal arteritis and thrombosis of the central retinal artery.** Klin. Monatsbl. f. Augenh. **121**: 80-81, 1952.

The author adds two cases from Sweden to this now well known association. Frederick C. Blodi.

François, Jules. **Electro-retinography in peripheral and central tapeto-retinal degenerations.** Ann. d'ocul. **185**:842-856, Oct., 1952.

In this study of 35 cases the electroretinograph of Karpe and the electrocardiograph of Elmquist were used. The latter was modified by using the active electrode on the cornea by means of a contact glass, the indifferent electrode on the temple, and the neutralizing electrode on the lids. The electroretinogram is absent in the peripheral form of primary retinal pigmentary degeneration and in other retinoses accompanied by night blindness; it is positive but subnormal in secondary pigmentary retinopathies especially during the early stages. The differential diagnosis in doubtful cases as well as the potential risks involved to unborn children may be facilitated by this test. The electroretinogram is positive, either normal or subnormal, in the macular degeneration of Stargardt and in other central tapeto-retinopathies, and positive but subnormal in generalized tapeto-retinopathies of the Sorsby type (inflammatory). (7 figures, 12 references) Chas. A. Bahn.

Jaeger, A. **Retinal veins with arterial blood.** Klin. Monatsbl. f. Augenh. **121**:84-85, 1952.

In a case of pseudotumor of the macula two veins carried arterial blood because of arteriovenous anastomoses. Frederick C. Blodi.

Karpe, G., and Rendahl, I. **The clinical electroretinogram. The electroretinogram in detachment of the retina.** Acta ophth. **30**:303-317, 1952.

Of 111 eyes with retinal detachment, both long-standing and recent, and of various etiology, all but seven had subnormal or extinguished electroretinograms. The height of the b-wave provides some measure of the functional capacity of the retina. In retinal detachment the amount by which the b-wave is decreased appears to depend on two factors. The larger the area of detachment and the poorer the general condition or vitality of the retina, the smaller does the b-wave become. That there is a relationship between vitality of the retina and the size of the b-wave appears to be indicated by the successful operations in cases of detachment with bigger b-waves, and failure of the retina to reattach in those with smaller b-waves. This relationship may be of practical value in deciding whether to operate in a given case or not. In addition to an association between the b-wave and the surgical prognosis, there appears to be an association between a subnormal electroretinogram and a tendency to detachment in the apparently normal eye in cases of unilateral detachment. This finding lends support to the theory of a predisposition to detachment. In some cases of unilateral detachment the normal eye with a poor electroretinogram developed a detachment later. It is believed that the electroretinogram may thus be utilized for an objective measurement of a predisposition to detachment. (8 figures, 5 references) Ray K. Daily.

Mackensen, G. **Fundus changes in Boeck's sarcoid.** Klin. Monatsbl. f. Augenh. **121**:51-63, 1952.

A 21-year-old woman who had a generalized lymphadenopathy showed bilateral proliferative retinal foci. The tuberculin test was negative in the con-

centration 1:100 and on the basis of a biopsy of one of the lymph nodes the diagnosis of Boeck's sarcoid was made. The retinal lesion showed a focal reaction after another tuberculin test and finally healed, leaving the picture of a retinal periphlebitis. (6 figures, 33 references) Frederick C. Blodi.

Magitot, A. **Idiopathic retinal detachment and its operative simplification.** Ann. d'ocul. **185**:857-865, Oct., 1952.

In 95 percent of so-called idiopathic retinal detachments, the primary lesion is a local specific degenerative disease in the pigment epithelium. The pigment epithelium has a glandular function and its secretions not only play an important part in the transformation of light into sight but also in the metabolism of the retina, especially its external layers. In retinal detachment this secretion is increased in quantity and altered in quality with resulting tendency to fissure and cyst formation as well as increased fluidity of the adjoining vitreous. Ultimately holes result. The subretinal fluid is a mixture of altered secretion of the pigment epithelium and degenerated vitreous. Galvanocautery with a fine point is preferred to diathermy because of its lower cost and greater ease of manipulation. The operative technique advised is that generally employed. Control of the operation by ophthalmoscopic inspection and prolonged bed rest are less important than the literature suggests. (1 figure)

Chas. A. Bahn.

Nover, Arno. **Endocrinogenic angiospastic retinitis.** Klin. Monatsbl. f. Augenh. **121**:40-46, 1952.

A 32-year-old patient with a severe angiospastic retinopathy had Cushing's disease six years previously but was cured after deep X-ray therapy. As the blood pressure was normal, the retinopathy was thought to be on an endocrine basis. (1 table, 33 references)

Frederick C. Blodi.

Riehm, W. **Central serous chorioretinitis.** Klin. Monatsbl. f. Augenh. **121**: 76-78, 1952.

The author describes an atypical case of this disease. In each eye there was a homogenous yellow exudate in the macula which had a sharp horizontal border above. After two and one-half years the exudate and all the symptoms disappeared. Frederick C. Blodi.

Roveda, José Maria. **Arteriovenous crossings and the prethrombosis sign of Paul Bonnet.** Arch. oftal. Buenos Aires. **27**:109-115, March, 1952.

Since 1934 Paul Bonnet has published several articles stressing the importance of the compression of the retinal vein by a crossing artery as a sign of prethrombosis of the vein. Bonnet also points out that Foster-Moore had said in 1924 that the immediate cause of a venous thrombosis should be sought in the crossing of a sclerosed artery. Several case reports with eight excellent fundus pictures serve to illustrate this occurrence.

Joseph I. Pascal.

Roveda, J. M., and Popi, M. **Retinochoroiditis of Jensen and tuberculous periarteritis.** Arch. oftal. Buenos Aires **27**:3-9, Jan., 1952.

The case presented here shows the picture of a retinochoroiditis of the Jensen type, combined with periarterial retinal lesions. This supports the hypothesis of Jensen that the basis of this disease is a disturbance in the vascular system. The authors point out that the disease picture originally described by Jensen should not be confused with other forms of retinochoroiditis. Joseph I. Pascal.

Sautter, H. **Peristatic phenomena in the fundus.** Klin. Monatsbl. f. Augenh. **121**:34-40, 1952.

Peristasis (Ricker) is a functional status of the vessels during which an arterial constriction causes dilation of the terminal branches. This is preceded by a generalized vascular constriction which may appear in the fundus as an ephemeral edema, exudate around the vessels, hemorrhages, and dishoric foci. (2 color plates, 2 figures) Frederick C. Blodi.

Siebeck, Robert. **Traumatic damage to the retinal vessel in juvenile hypertension.** Klin. Monatsbl. f. Augenh. **121**:46-50, 1952.

This is a report on two young patients with hypertension who developed a unilateral hypertonic fundus in the eye that was injured by a severe contusion. The author assumes that the originally reflex angiospasm became permanent by subsequent structural changes. (19 references) Frederick C. Blodi.

Wilson, Fred M. **Ophthalmoscopic evaluation of the hypertensive patient.** J. Kansas M. Soc. **53**:225-229, May, 1952.

The most useful methods of grading and classification of retinal hypertensive changes, the Wagener-Clay-Gipner method and the Keith-Wagner-Barker grading are discussed in detail. The author feels that each method fulfills a different function; the former is detailed and exact, the latter more practical. These methods may be used individually or in combination. Certain clinical applications of detailed eyeground observation and a brief differential diagnosis of certain hypertensive changes are mentioned. (1 figure, 3 references) James V. Bolger.

12

OPTIC NERVE AND CHIASM

Asayama, R., Goto, Y., Makino, Y., and Adachi, H. **Anterior pituitary hormone in the treatment of optic nerve atrophy caused by tabes dorsalis.** Acta Soc. ophth. Japan **56**:1323-1329, Dec., 1952.

An injection of anterior pituitary hormone and vitamin B_1 into the carotid artery for 7 to 175 days showed a favourable effect in four of the nine cases of simple atrophy of the optic nerve caused by tabes. Some improvements were brought about in vision, the visual field and color sense. Yukihiko Mitsui.

Hisatomi, Y. **Study of power of accommodation in cases of nutritional chronic retrobulbar optic neuritis.** Acta Soc. ophth. Japan **56**:1369-1382, Dec., 1952.

In 82 cases of chronic retrobulbar optic neuritis due to a deficiency in vitamin B_1, the power of accommodation was measured. In patients, 15 to 20 years old, a 1.5 to 2.5-diopter reduction in the accommodative power was recognized, in those 20 to 30 years old, a 0.5 to 1.5 diopter reduction. (9 figures, 5 tables) Yukihiko Mitsui.

Korff, Jürgen. **Angioma of the optic nerve.** Klin. Monatsbl. f. Augenh. **121**:68-70, 1952.

This rare tumor of the optic sheaths clinically simulated meningioma of the sphenoid ridge. On operation a large tumor of the optic nerve was found which extended into the cranium and was continuous with the globe. Histologic examination revealed a capillary hemangioma. (3 figures, 4 references) Frederick C. Blodi.

Tosi, B., and Gayoso, C. B. **Posthemorrhagic optic atrophy.** Arch. oftal. Buenos Aires **27**:124-127, March, 1952.

A 51-year-old man was always well until three years previously when he had an intestinal hemorrhage which left him unconscious for 22 days. A month and a half after leaving the hospital he noticed that his sight was deteriorating. He now showed bilateral postneuritic optic

atrophy, with vision of 2/10 in each eye. The field of vision was greatly reduced, with a tendency to binasal hemianopsia. The optic atrophy is ascribed by some to an unknown toxin that may be liberated by the pathologic process which caused the original excessive hemorrhage and also to ischemia of the retina due to excessive loss of blood.

Joseph I. Pascal.

Yonemura, Taizo. **Applicability of Ferry-Porter's law of flicker test in retrobulbar neuritis.** Acta Soc. ophth. Japan **56**:1335-1339, Dec., 1952.

In three cases of retrobulbar neuritis with central scotoma, the relative summation of the critical fusion frequency of flicker was proved to be smaller than that in normal eyes, therefore, the Ferry-Porter law is not applicable here. The same thing was demonstrated in congenital total achromatopsia. The author considers such a disturbance to be due not only to a disorder of the light-perceiving layer of the retina but also to that of the visual paths. Yukihiko Mitsui.

13

NEURO-OPHTHALMOLOGY

Bedrossian, E. Howard. **Raeder's syndrome.** A.M.A. Arch. Ophth. **48**:620-623, Nov., 1952.

A patient with Raeder's syndrome recovered after the removal of an abscessed tooth. Raeder's syndrome is like Horner's syndrome without vasomotor or trophic disturbances and is the result of coincident sympathetic disturbance and a lesion of the trigeminal nerve. The completeness of a Horner's syndrome depends upon the site of involvement of the sympathetic fibers. A complete Horner's syndrome occurs when the site is cervical, but is incomplete and often accompanied by other neurological signs when the site is spinal. When the site is paratrigeminal, Raeder's syndrome results. (2 figures, 3 references) George S. Tyner.

Fanta, H., and Reisner, H. **Argyll Robertson pupil with suprasellar tumor.** Klin. Monatsbl. f. Augenh. **121**:63-68, 1952.

This patient had a typical Argyll Robertson pupil. The cause was a craniopharyngioma and, after excision of the tumor, the pupillary reactions became nearly normal, showing that an Argyll Robertson pupil is not always pathognomonic for a syphilitic infection. (10 references) Frederick C. Blodi.

Giller, H., and Cogan, D. G. **Papilledema as the outstanding sign in meningeal hydrops.** A.M.A. Arch. Ophth. **48**:557-566, Nov., 1952.

Meningeal hydrops is a clinical entity in which increased intracranial pressure is present without any space-taking lesion or other obvious cause. The presenting signs and symptoms are blurred vision, headache and bilateral symmetrical papilledema. The prognosis is almost always favorable. The most common known cause is thrombosis of the dural venous sinuses. (19 references)

George S. Tyner.

Hauser, M. H., and Gass, H. **Optic nerve pressure of aneurysm relieved by decompression of optic nerve.** A.M.A. Arch. Ophth. **48**:627-631, Nov., 1952.

In a 34-year-old white woman an aneurysm of the circle of Willis caused rapid loss of vision in her one useful eye. Restoration of visual function was obtained by decompression of the optic nerve. The roof of the right optic foramen was removed through a right frontal craniotomy. The position of the aneurysm precluded ligation. (3 figures)

George S. Tyner.

Lawrence, G. Allen. **Bilateral occipital-lobe infarction simulating retrobulbar optic neuritis.** A.M.A. Arch. Ophth. **48**: 602-604, Nov., 1952.

A patient is reported who showed a

bilateral pericentral scotoma proved by autopsy to be caused by symmetrical occipital lobe thrombosis. It was believed by the author that treatment with corticotropin may have been a contributory cause of death by increasing the tendency toward thrombosis. (2 references)

George S. Tyner.

Malbran, J. L., Sitler, R. and Insausti, T. **Homonymous hemianopic paracentral scotoma.** Arch. oftal. Buenos Aires **27**: 193-212, May, 1952.

The authors present an extensive résumé of the literature covering the homonymous, hemianopic paracentral scotomata of spontaneous origin. In the patient presented, a 33-year-old woman, the author at first suspected that antecedent trauma to the occipital region had some relation to the appearance of the scotoma, but a study of the patient made him discard this possibility as well as the possibility of a vascular or neoplastic lesion. The complete recovery suggests the hypothesis of a demyelinizing process, possibly multiple sclerosis. The probable localization of the lesion in this patient is in the optic pathways anterior to the optic radiations. (12 figures)

Joseph I. Pascal.

Pshenichnova, A. A., **Observations of homonymous hemianopsia in hypertension.** Vestnik Oftal. **29**:28-33, May-June, 1950.

The author discusses homonymous hemianopsia occurring in hypertension. The study of 17 cases of hemianopsia is presented, in 15 of which the higher centers were affected and in two the optic tract; in 11 of them hemianopsia was the only neurological sign. The other four had: 1. sensory aphasia, 2. alexia and agraphia, 3. hemiparesis, and 4. a disturbance of the extrapyramidal area. The onset of hemianopsia was sudden and coupled with the loss of central vision which recovered in a short time. Of the patients with lesions of higher nervous centers four had retinal changes. The extent and form of hemianopsia was varied. In four cases it had decreased on the second examination and was stationary in seven.

Sylvan Brandon.

Rieger, H. **The Pathology of the ciliary ganglion.** Klin. Monatsbl. f. Augenh. **120**:337-346, 1952.

The author reports two cases of unilateral internal ophthalmoplegia combined with retrobulbar neuritis on the same side. He assumes that an inflammatory or toxic lesion of the ciliary ganglion was the cause of this condition.

Frederick C. Blodi.

Rucker, C. Wilbur. **Neuro-ophthalmology.** A.M.A. Arch. Ophth. **48**:639-656, Nov., 1952.

The principal published papers on the subject during the past year are reviewed. The subjects covered are perimetry, optic nerves and chiasm, papilledema, pupils, ocular movements and related syndromes. (102 references)

George S. Tyner.

Spalding, J. M. K. **Wounds of the visual pathway.** J. Neurol. Neurosurg. & Psychiat. **15**:169-183, Aug., 1952.

A series of 958 cases of penetrating head injury, mainly gunshot wounds, has been reviewed to determine what light they throw on the anatomy of the striate cortex (area 17). In 188 of these there was a visual field defect attributable to injury to the visual radiation or striate cortex. In 72 cases the injury primarily affected the striate cortex, and of these characteristic examples are presented and their anatomic significance discussed.

Central ("macular") vision is represented unilaterally. The horizontal meridian of the visual field is represented in the floor of the calcarine fissure. The extent of striate cortex devoted to central vision is defined. Central vision within the 8° to 10° circumference (i.e. macular

vision) is represented on that part of the striate cortex which faces posteriorly or postero-medially. The remainder, which faces medially, represents vision more peripheral than 10° from the fixation point. The lips of the calcarine fissure at the point where the striate cortex on the medial surface of the hemisphere becomes continuous with that on the postero-medial surface represents the points 8° from the fixation point and 30° from the vertical meridian. (11 figures, 16 references) Theodore M. Shapira.

Straub, Wolfgang. **Nasal hemianopsia.** Acta ophth. **30**:229-251, 1952.

Four cases are reported in three of which the diagnosis was verified anatomically. The first patient was treated by decompression. A diagnosis of malignant neoplasm originating in the third ventricle was made and was not verified by autopsy. The second case was that of a left sided parasagittal meningioma originating in the falx cerebri. The third patient had a carcimoma in the nasopharynx which invaded the cranium through the sphenoid sinuses. The fourth patient had an angioma of the medulla oblongata, with blocking of the ventricles and internal hydrocephalus. All four patients gave a history of transient attacks of loss of vision; Straub urges that in daily practice such vague symptoms be thoroughly investigated. All four patients first consulted an ophthalmologist. The ocular disturbance was bilateral from the beginning. The field defects were asymmetrical as to size and position. Three patients had bilateral choked discs which passed into optic atrophy, and one had an optic atrophy when first seen. X-ray examination revealed sellar changes in all. The response to Roentgen therapy was very different in the four patients. In the first patient vision and visual fields deteriorated rapidly; in the third there was a marked improvement in the ocular symptoms, which lasted for a considerable length of time. Obviously binasal hemianopsia is not a reliable localizing sign. (13 figures, 41 references)

Ray K. Daily.

Wagman, Ora H. **Sudden blindness due to intracranial vascular accident.** New England J. Med. **247**:7-10, July 3, 1952.

In a 77-year-old man sudden total and permanent blindness was the result of bilateral infarction of the visual cortex, which resulted in simultaneous bilateral homonymous hemianopia without sparing of the macula. Basic anatomy, pathogenesis and differential diagnosis are discussed. (4 figures, 9 references)

James V. Bolger.

14

EYEBALL, ORBIT, SINUSES

Hoegel, L. **Intermittent exophthalmos and amaurosis.** Klin. Monatsbl. f. Augenh. **120**:346, 1952.

A 60-year-old woman who had attacks of intermittent exophthalmos became blind after an episode that lasted for several hours. The author explains the amaurosis as a mechanical effect of venous pressure on the optic nerve.

Frederick C. Blodi.

15

EYELIDS, LACRIMAL APPARATUS

Desvignes, P., and Sadoughi, G. **A case of reticulolymphosarcoma of the lacrimal sac.** Arch. d'opht. **12**:524-526, 1952.

The authors note the rarity of tumors in the lacrimal sac and their late recognition when they do occur. They report the case of man of 36 years with a bean-sized tumor of the right lacrimal sac. There had been symptoms of conjunctivitis and tearing for several years previously and probings had been unsuccessful. The sac was excised and on section was found to be invaded by a reticulosarcoma. Postoperative radiation was employed and no further extension of the tumor had been noted at the time of writing. The various

clinical signs of lacrimal sac tumor are considered in detail and the conclusion is reached that a tumor should be suspected in the presence of abnormal hardness of the sac and on the occurrence of spontaneous bleeding. Since certain tumors have masqueraded as ordinary mucoceles, the importance of microscopic examination is stressed. (2 figures, 8 references)

Phillips Thygeson.

Fox, Sidney A. **Correction of senile entropion.** A.M.A. Arch. Ophth. **48**:624-626, Nov., 1952.

The author presents a modification of the technique of his operation for senile entropion originally reported in the October, 1951 issue of the Archives of Ophthalmology. In the modification, lid splitting is avoided and the position of the temporal skin-muscle excision is changed. The operation consists essentially of excision of a triangular piece of tarsus and conjunctiva with apex toward the lid margin, combined with excision of a vertically elliptical portion of the skin and orbicularis muscle temporal to the external canthus. The skin and muscle are separated from the tarsus between the two excisions by scissors dissection to allow the lips of the wounds to be easily approximated. (1 figure, 2 references)

George S. Tyner.

Hudelo, A., and Mergier, J. **The study of the lacrimal pH in the function of local and general states.** Ann. d'ocul. **185**:764-771, Sept., 1952.

The ionic equilibrium of the tears is of especial interest because it is related to cellular and intracellular secretion and metabolism both in health and disease. The pH of the tears both normally and abnormally varies widely in different individuals and in the same individual in the two eyes and at different times. Tests made with blotting paper (Schirmer) are not accurate because of injury to the conjunctiva. If such tests are necessary because better equipment is lacking, cigarette paper should be used. The authors have invented an electric method for determining the pH which they consider accurate. After the instillation of ordinary acid or alkaline collyria, a primary reaction is followed by a secondary reaction of the opposite type, both completed within ten minutes. In a subsequent contribution, the authors will discuss the pathologic and therapeutic applications of their method. (3 figures)

Chas. A. Bahn.

Morgan, A. D., and Raven, R. W. **Sjögren's syndrome: a general disease.** Brit. J. Surg. **40**:154-162, Sept., 1952.

Two cases of Sjögren's syndrome are presented and discussed from the standpoint of etiology (estrogenic deficiency, chronic infection, fibrosis, and the neurotropic factors). Ocular and nonocular manifestations such as xerostomia, parotid swellings, polyarthritis of the rheumatoid type and dryness of the skin and mucous membranes occur. The microscopic pathology is presented in detail. (16 figures, 47 references)

Irwin E. Gaynon.

Pavišič, Z. **Tarsoplasty for entropion and trichiasis.** Ophthalmologica **123**:35-38, July, 1952.

For the "worst degrees" of cicatricial entropion and trichiasis the author recommends a procedure consisting of 1. turning the lid border away from the globe by a tarsotomy made from the conjunctival side along the entire subtarsal sulcus, 2. fastening the lid border in its new position by appropriate sutures, and 3. placing a strip of buccal mucous membrane between the main body and the everted distal strip of the tarsus. (5 figures, 11 references)

Peter C. Kronfeld.

Rocco, Alberto. **A case of button fever.** Rassegna ital. d'ottal. **21**:290-297, July-Aug., 1952.

Button fever is caused by a Rickettsia. A 35-year-old woman received a spurt of purulent fluid in the right eye from the sore of a dog infected with tick. The eye was immediately and thoroughly washed out. Two days later, symptoms of malaise, suffocation, dizziness and nausea appeared. The eye became injected, a nodule formed on the lower lid margin, and the preauricular gland became swollen. Penicillin, chloromycetin and aureomycin were applied. Recovery was complete.

Eugene M. Blake.

Roveda, José Maria. **Dacryotunnelization.** Arch. oftal. Buenos Aires **27**:32-35, Jan., 1952.

Roveda describes an operation suitable for patients with deformities after old ocular trauma involving the lacrimal apparatus, the lids and the globe, with excessive, excoriating tearing. The author describes his operation as a dacryolacorhinostomy. (5 figures)

Joseph I. Pascal.

Strampelli, B. **Shortening of the orbicularis muscle by torsion for correction of spastic entropion.** Boll. d'ocul. **31**:496-500, Aug., 1952.

After an L-shaped incision parallel to the lower lid border and extending vertically down from below the temporal lid angle, a triangular skin flap is undermined to expose the orbicularis muscle. Some fibers of this muscle are separated and, using a short needle, twisted several times. The muscle roll thus formed is now fixed to the upper and lower skin wound lip. The procedure was successful in three cases of spastic entropion. (8 figures)

K. W. Ascher.

Victoria, V., and Gordillo, C. H. **Lagophthalmos and leprosy.** Arch. oftal. Buenos Aires **27**:105-108, March, 1952.

A 44-year-old man is described who developed complete lagophthalmos, with paralysis of the orbicularis muscles in both eyes as a result of having contracted leprosy two years before. The lids in both eyes are infiltrated, but pliable. There is slight ectropion, with everted lacrimal puncta and very marked Bell's phenomenon. The eyes are normal in other respects.

Joseph I. Pascal.

Vidal, F., and Weil, B. **Lipid blepharosis.** Ann. d'ocul. **185**:778-783, Sept., 1952.

Seborrheic blepharitis usually is the result of two pathologic processes; abnormal lipid secretion and infection. Lipid blepharosis applies only to the former. The lid margins become thicker, grayish yellow, oily to touch or pressure, easily congested and infected. Dilatation of the Meibomian gland ducts and lacrimal puncta as well as tissue hyperplasia are frequent sequellae. The oily secretion of the Meibomian glands which consists largely of oleic acid, normally forms an emulsion with the secretion of the sweat glands. In lipid blepharosis the glands secrete additional chains of carbon atoms forming the more paraffin-like stearic acid compounds which tend to block the gland ducts and to cause a low grade inflammatory reaction in the surrounding tissues. The skin of the face is usually also involved. In lipid blepharosis the bodily androgens are usually increased and the estrogens decreased. Lipid blepharosis is usually an ocular manifestation of faulty bodily lipid metabolism and of vagotonia with its allergic hypersensitivity and pain. (10 references)

Chas. A. Bahn.

Vidal, F., and Weil, B. A. **Sebaceous blepharosis.** Arch. oftal. Buenos Aires **27**: 229-233, May, 1952.

The authors describe a clinical picture of the precorneal space with manifestations of hypersecretion by the sebaceous glands at the lid border. They label the condition "sebaceous (fatty) blepharosis."

Joseph I. Pascal.

16
TUMORS

Cook, C. A. **The life history of pigmented naevi.** Tr. Ophth. Soc. U. Kingdom **71**:257-263, 1951.

The majority of naevi are congenital in origin and have been divided by Allen (1949) into three main subdivisions. 1. Junctional naevi are characterized by the presence of intraepidermal cell nests broken loose from adjacent epidermal cells, and occur in the basal epidermal layers at the junction of epidermis and dermis and the overlying stratum mucosum may also be involved. 2. In dermal naevi naevus cells are arranged as cell nests diffusely scattered throughout the dermal layers. 3. Compound naevi consist of a combination of the intraepithelial and dermal forms. The growth of a naevus in characterized by early proliferative, adult quiescent, and senile retrogressive stages. (4 figures, 7 references) Beulah Cushman.

Fontana, G. **A case of basal cell epithelioma of the lid progressing rapidly.** Gior. ital. oftal. **5**:265-270, July-Aug., 1952.

A small nodule of the upper lid in a man, 64 years of age, developed into a tumor the size of a walnut within six months. It had invaded the lower lid and the orbit. Biopsy showed an anaplastic basal cell epithelioma. Exenteration of the orbit was performed and eight months later no metastases were found. (3 figures, 18 references) John J. Stern.

François, J., and Rabaey, M. **Primary tumors of the optic nerve.** Acta ophth. **30**:203-221, 1952.

A primary glioma and two meningioblastomas of the optic nerve are described. The meningioblastomas originated from the sheaths of the intraorbital optic nerve. The histologic structure of the tumors, illustrated with photomicrographs, shows that one meningioblastoma consisted of spindle-shaped cells grouped in whirled bundles, in an alveolar arrangement. In the second case the tumor consisted largely of very long cells of fibrillar appearance, with areas of myxomatous tissue and other areas of alveolar structure. (18 photomicrographs, 4 references) Ray K. Daily.

Miyazaki, Shigeo. **Metastasis of mammary cancer in the orbit.** Acta Soc. Ophth. Japan **56**:1198-1201, Oct., 1952.

Metastasis to the right orbit was noted in a 46-year-old woman five months after the extirpation of a mammary cancer of the left side. No other metastases were observed. Yukihiko Mitsui.

Provasi, Giuseppe. **Palpebral hydroadenoma.** Gior. ital. oftal. **55**:335-347, July-Aug., 1952.

Three cases are reported with histologic details. Provasi believes that hydroadenoma originates in the sweat glands and discusses the possibility that an endocrine dysfunction may be the etiological factor. (4 figures, 19 references) John J. Stern.

Saebo, J. A. **Hippel-Lindau's disease.** Acta ophth. **30**:129-154, 1952.

Five cases of Hippel-Lindau's disease occurred in one family. The eye symptoms became manifest after the age of 20 years. One patient, 32 years old, had no subjective symptoms, but a fundus picture characteristic of the disease. Eight children of the affected parents, all under ten years of age, had no evidence of the disease. The disease is progressive and ends in blindness or death at a relatively young age. Transmission is dominant. Therapeutic results are discouraging. One patient treated by diathermycoagulation had an immediate satisfactory result, but later the disease spread over the fundus and led to blindness. On another patient diathermycoagulation was followed by a retinal detachment. The removal of a cerebellar

tumor in one patient was followed by an excellent result. The disease was bilateral in these cases and there was a combination of a retinal and cerebellar angiomatosis. The changes in the retinal vessels are typical, and are found in the two vessels leading to the angiomatous tumor. The veins are enlarged and tortuous, the arteries enlarged and irregular in caliber. The two vessels are alike in color. Degenerative or atrophic areas may be seen near the tumor or spreading over the fundus. Small whitish patches similar to retinitis circinata also occur. (5 figures, 1 table, 34 references) Ray K. Daily.

Uihlein, A., and Miller, R. H. **Unusual orbital tumors: report of three cases.** Proc. Stf. Meet. Mayo Cl. **27**:402-407, Oct. 8, 1952.

The authors report three cases of unusual intraorbital tumor removed by the transcranial route. The tumors were a solitary neurofibroma associated with glaucoma, a hematoma, and a cylindroma of the lacrimal gland. (3 figures)
Irwin E. Gaynon.

17
INJURIES

Musial, Albin. **Two cases of traumatic rupture of the optic nerve.** Klinika Oczna **22**:153-160, 1952.

Two cases of traumatic evulsion of the eyeball are described. In one of them the injury was probably self inflicted. There was no rupture of the globe and very little damage to the conjunctiva. The author discusses the mechanism of evulsion of the eyeball. (2 figures) Sylvan Brandon.

Tikhomirov, P. E., **Chemical analysis of the aqueous for the presence of copper.** Vestnik Oftal. **29**:9-13, Jan.-Feb., 1950.

On many occasions the character of an intraocular foreign body cannot be established, particularly if it is nonmagnetic. The author describes a method of chemical analysis for copper by which one can demonstrate the presence of minute amounts of this metal in the aqueous. The technique of this analysis is presented. Experiments on rabbits proved the feasibility and applicability of this analysis. The same technique used on four patients proved its clinical value. Foreign bodies, nonmagnetic or not demonstrable by X-ray examination, were found and removed. Sylvan Brandon.

Vogt, L. G. **Retrobulbar gunshot without damage to the eye.** Klin. Monatsbl. f. Augenh. **120**:539-543, 1952.

A gunshot bullet after traversing the right orbit reached the left orbit and remained in the retrobulbar space without any damage to the visual organ. (3 figures, 6 references) Frederick C. Blodi.

18
SYSTEMIC DISEASE AND PARASITES

Barnes, H. D., and Boshoff, P. H. **Ocular lesions in patients with porphyria.** A.M.A. Arch. Ophth. **48**:567-580, Nov., 1952.

Small amounts of porphyrins appear in the urine of normal subjects. Increased amounts give a red color to the urine and are seen occasionally in individuals in association with a bullous skin eruption, or abdominal colic, or after the use of some drugs, or exposure to certain toxic substances. An acute and chronic form of porphyria occurs.

Eighty-four patients with porphyria are reported with a description of associated ocular signs, particularly hitherto undescribed fundus changes. The principal external ocular changes resembled those of pemphigus or trachoma with bullous lesions of the lids and conjunctiva followed by scarring. Corneal scaring and scleral thinning also occur. The principal fundus lesions were seen during cerebral episodes of acute porphyria. They are of two types, cotton-wool patches and peripheral areas of edema in the superficial

nerve fiber layer. Residual pigment deposits and yellowish plaques were seen at the site of the acute lesions. (3 tables, 8 figures, 16 references)

George S. Tyner.

Burk, Martin. **Ocular lesion in arteritis temporalis.** Klin. Monatsbl. f. Augenh. 120:273-278, 1952.

The author reports another case from Hamburg (most of the German cases were reported from that city). The pain along the temporal artery was mild, but a biopsy proved the diagnosis. There was a unilateral occlusion of the central retinal artery. Fever and increased ESR were, as usually, present. (3 figures, 1 color plate, 15 references) Frederick C. Blodi.

Cordes, Frederick C. **The diabetic: his visual prognosis.** A.M.A. Arch. Ophth. 48:531-556, Nov., 1952.

The auther briefly reviews pertinent general information about diabetes. The bulk of the paper is a résumé of the literature on the ocular complications of diabetes. There are two types of diabetes, the hyperfunctional type in which the pancreas is unable to deal with a hyperproduction of sugar caused by hypophyseal stimulation and the rarer hypofunctional type which is primarily a pancreatic disease. Approximately 70 percent of diabetics are in the former group and 30 percent in the latter.

The disease is probably transmitted as a simple Mendelian recessive. Obesity is a predisposing factor. The life expectancy of diabetics is normal with modern treatment unless contracted in childhood or adolescence. The principal complications are cardiovascular-renal in type and thus affect the eye. Twenty percent of diabetics show weakness of accommodation. This is seen most often in the younger patients. Alterations in refraction include marked changes in astigmatic errors as well as in hyperopia and myopia. Berry-like conjunctival aneurysms occur four times as frequently in diabetics as in nondiabetics. The visual prognosis in diabetics with rubeosis iridis is poor. If glaucoma occurs with rubeosis iridis, treatment by any means is discouraging. Cyclodiathermy is probably the most successful treatment and if this fails enucleation is the only recourse. The operative procedure of choice in true diabetic cataracts (snow-flake type) is linear extraction. In older diabetics with senile cataracts the possibility of hemorrhage is increased by 20 percent if an iridectomy is performed at the time of lens extraction. It is also important to refrain from giving insulin in large doses on the day of the operation.

The development of retinopathy is related to the duration of the disease rather than the severity. Reports are accumulating, however, which indicate that rigid control of diabetes over a long period of time may favorably influence the visual prognosis. The best treatment is adequate control of the diabetes. The efficacy of testosterone therapy has not yet been adequately evaluated. Rutin may be more valuable in the control of vitreous hemorrhage than in control of the retinopathy.

In pregnancy, the prognosis for a normal gestation is 47 percent better in women with little or no retinopathy than in those with retinopathy. In general, the presence of retinopathy is of no prognostic value in life expectancy. The visual prognosis is poor in diabetics who have had the disease for twenty years or more. Hence juvenile diabetics have the poorest visual prognosis since their survival time is longer than those who acquire the disease in later life. (8 figures, 128 references) George S. Tyner.

Hartmann, Karl. **Ocular complications in Still's disease.** Klin. Monatsbl. f. Augenh. 121:216-221, 1952.

The author reports another case of Still's infantile polyarthritis associated with bilateral band keratopathy, iritis

and complicated cataract. (5 figures, 18 references) Frederick C. Blodi.

Jendralski, Hans J. **Recovery from diabetes insipidus after bilateral cataract extraction.** Klin. Monatsbl. f. Augenh. **121**: 86-90, 1952.

A surprising cure of a longstanding diabetes insipidus occurred after bilateral cataract extraction. The author assumes that the increased light perception caused a change on the hypothalamic centers. (15 references) Frederick C. Blodi.

Krawczyk, Zofia. **A case of ocular tularemia.** Klinika Oczna **22**:161-164, 1952.

Ocular tularemia occurred in a woman, 23 years of age. On the first examination the left eye presented symptoms of Parinaud's conjunctivitis. The membrane was located on the nasal side of the eye. The periauricular gland was swollen and tender. There were general febrile symptoms. Under local treatment there was slight improvement. During three weeks of observation all tests for tuberculosis were negative. The patient had handled a rabbit two months before the beginning of the disease. An intradermal test for tularemia was highly positive. The patient was cured by repeated injections of penicillin. (20 references) Sylvan Brandon.

Lampen, H. **The operative risk in vascular hypertension.** Klin. Monatsbl. f. Augenh. **121**:139-147, 1952.

The author, an internist, gives a short survey of some of the old, well-known theories of vascular hypertension. He discusses the management of a patient with hypertension before and after operation. (21 references) Frederick C. Blodi.

Mark, Robert E. **The importance of hypertension in vascular and renal diseases.** Klin. Monatsbl. f. Augenh. **120**:278-289, 1952.

This is a short survey on the various forms of hypertension given by an internist for ophthalmologists. The retinal involvement is of prognostic importance especially when evaluated together with the results of the urine analysis and renal function tests. The author follows Volhard's scheme closely. (5 figures, 7 references) Frederick C. Blodi.

Pages, R. **Ocular complications of relapsing fever.** Semaine d'hôp. Paris **94**: 3824-3827, Dec., 1952.

Ocular and neuromeningeal complications are common in relapsing fever especially that caused by tics and all practitioners in Africa should be aware of them. Uveitis is a later complication, rarely developing before the third week and occurs in 20 to 30 percent of cases. The aqueous is cloudy with deposits on Descemet's membrane and often the vitreous is opaque. There may be iridochoroiditis with much pain. Iris and ciliary vessels are injected and there may be retrolental opacities. Lesions of the optic nerve are less common, but when they do occur, they may permanently affect vision. Visual acuity and the field are lessened. There may be retrobulbar neuritis and scotoma. Primary optic atrophy results. When meningoencephalitis has developed, a spread along the nerve sheath may lead to papillitis and increased ocular tension. Toxic neuritis is frequently seen as the result of treatment with arsenic, especially the pentavalent arsenicals. (9 references) B. T. Haessler.

Piper, H. F. **Ocular diseases in malnourished patients.** Klin. Monatsbl. f. Augenh. **120**:357-373, 1952.

The author analyzes the eye diseases found in 87 prisoners of war returning from Russia. No disease was present that could be ascribed specifically to undernourishment. The incidence of eye diseases was, however, high.

Frederick C. Blodi.

Savin, L. H. **Ocular disease and arthritis caused by the same organism. Tr.**

Ophth. Soc. U. Kingdom 71:133-146, 1951.

The author draws attention to the fact that ophthalmia neonatorum can be followed by gonococcal arthritis and pneumococcal conjunctivitis by pneumococcal arthritis. Many patients with brucellosis develop joint pains with swelling and effusion. The diagnosis is made by eliminating other causes and eliciting positive serologic reactions or other evidence of sensitivity. Arthritis is a rather infrequent complication of bacillary dysentery, usually occurring after the acute bowel symptoms have subsided. Joint tuberculosis has often been associated with such eye diseases as phlyctenulosis, episcleritis, scleritis, iridocyclitis, choroiditis and miliary choroidal tubercles.

Gout is frequently associated with ocular complications. Of 12 patients, 6 had iridocyclitis, 3 episcleritis or scleritis and 1 each had marginal sclerosing keratitis, conjunctivitis, and secondary glaucoma with cataract. The iritis and uveitis frequently respond to small doses of colchicum. Cortisone may also be of value. Hemophilia is almost invariably associated with hemarthrosis especially of the knee joints. Scurvy in infants may also be accompanied by hemarthrosis. The rheumatoid arthritis group of diseases associated with iridocylitis include Still's disease, ankylosing spondylitis, psoriatic arthritis and Sjögren's syndrome.

The value of Cortisone or ACTH in the treatment of all of these diseases with the complicating eye conditions have been reported, although their use may cause a condition resembling Cushing's syndrome, and the distressing symptoms following its withdrawal are usually associated with relapses and severe depressions. (17 references)

Beulah Cushman.

Siegert, P. **Acute ischemia of the disc in temporal arteritis.** Klin. Monatsbl. f. Augenh. **120**:254-273, 1952.

The author discusses signs and symptoms of temporal arteritis. He tabulates all the cases reported in the German literature; 15 cases have occurred and approximately one-half had ocular signs. The ocular signs are of three kinds: 1. Acute ischemia of the retina may be caused by an occlusion of a central vessel. 2. There may be diffuse changes in the vascular system of the eye. 3. Acute ischemia of the disc was observed by the author in two patients with the typical signs of a temporal arteritis. The discs were swollen, edematous and pale. The visual impairment was sudden, severe and irreversible. This ophthalmoscopic picture is probably precipitated by pathologic changes in the vessels of the intrascleral part of the optic nerve. The author believes that these changes occur only in temporal arteritis and a third case which showed only an ischemia of the disc without any other signs was thought to present the primary ocular form of this disease. (10 figures, 1 color plate, 47 references)

Frederick C. Blodi.

Vidal, Flaminio. **Diminution of the protein content of the precorneal liquid.** Arch. oftal. Buenos Aires **27**:11-13, Jan., 1952.

The author describes the clinical picture of the precorneal chamber (the interpalpebral fissure) where there is a diminution in the protein content of the liquid on the surface of the cornea, combined with a general disturbance such as hypoproteinemia and a lowering of the albuminous fraction in the blood-serum. The condition responds well to local and general treatment with amino acids, especially sulphurous amino acids.

Joseph I. Pascal.

19

CONGENITAL DEFORMITIES, HEREDITY

Burk, M., and Northoff, F. **Ocular findings in blue babies.** Klin. Monatsbl. f. Augenh. **120**:351-357, 1952.

The ocular sign in 74 patients with

congenital heart defects are evaluated. The well-known cyanosis and tortuosity and dilatation of the vessels were found and correlated with the degree of general cyanosis and polycythemia.

Frederick C. Blodi.

François, F., and Rabney, M. **Histochemical examination of corneal dystrophy and hereditary study in a case of gargoylism (Hurler's disease).** Ann. d'ocul. **185**:784-804, Sept., 1952.

Hurler's disease (dysostosis multiplex) is apparently not a disease of lipid metabolism as is generally believed. The affected cells of the corneal stroma do not take specific stains for fat, glycogen, mucin, nor collagen. They contain a granular substance, are vacuolated and most abundant near Bowman's membrane which in the case reported was almost destroyed. These cells resemble phagocytes in structure and are present in other parts of the eye and body. Their chemical classification is not understood. Numerous cases in the literature are discussed to illustrate the authors' conclusions. Characteristic corneal opacities, gargoylism, enlargement of liver and spleen, and marked mental retardation were present in the five-year-old boy who is described in detail. The parents and great grandparents were blood relatives. Six cases of Hurler's or allied diseases occurred in six generations, hence the recessive mode of transmission. The histologic studies were made from a partial corneal transplant. (9 figures, 163 references)

Chas. A. Bahn.

Keizer, D. P. R. **A new form of congenital hereditary deafness (Waardenburg's syndrome).** Nederl. tijdschr. v. geneesk. **96**:41-2541, 1952.

In 1951 Waardenburg described a new form of irregularly dominant deafness. This syndrome consists of prominent nasal bones and root of the nose, hypertrophy of the medial parts of the eyebrows which are usually grown together, lateral displacement of the inner canthi of the eyes, while the external canthi are situated in their normal place, with a resulting shortening of the horizontal lacrimal fissure, a white lock of hair, partial or total heterochromia and deaf-mutism. The author describes three generations of a family in which one child is normal, four have deaf-mutism, two members have a congenital unilateral deafness and are gray-haired at an early age, and two have a heterochromia. In this family the pattern of inheritance of the congenital deafness is dominant and not-sex linked.

G. H. Jonkers.

Moller, P. M. **Another family with Von Hippel-Lindau's disease.** Acta ophth. **30**:155-164, 1952.

A pedigree containing five verified and four probable cases of Hippel-Lindau's disease is given. Of 40 members, 29 were examined. The first generation contains seven members, of whom one was probably affected with the disease. This patient had one eye enucleated, but because of death an examination was not possible. In the second generation, consisting of ten members, one member had retinal angiomatosis; another, who died suddenly, might have had a cerebellar lesion. In the third generation, three of 23 members had definite angiomatosis, and one a congenital cyst in the kidney. The heredity in these cases is irregularly dominant, with no sex linkage. (1 figure, 7 references)

Ray K. Daily.

Pitter, J., and Švejda, J. **The teratogenic effect of Roentgen radiation.** Ophthalmologica **123**:386-393, June, 1952.

Multiple congenital defects are reported in a two-year-old girl whose mother had had repeated fluoroscopic chest examinations during the 4th, 5th, and 6th month of pregnancy. (6 figures, 30 references)

Peter C. Kronfeld.

Straub, W., and Heberling, V. **The Laurence-Moon-Bardet-Biedl syndrome.** Klin. Monatsbl. f. Augenh. **120**:289-298, 1952.

The authors report five cases which occurred in three families. A recessive type of heredity is presumed. (6 figures, 20 references) Frederick C. Blodi.

de Vincentiis, Mario. **The report of a case of human teratology, and its contribution to the knowledge of the morphologic and organogenetic properties of the human optic vesicle.** Acta ophth. **30**:255-279, 1952.

The author describes in detail the clinical and pathologic data of an epibulbar cyst which was found to contain retinal and lens tissue in varying stages of development. He also reports his investigations in experimental embryology of the morphologic and organogenetic properties of the optic vesicle in embryos of amphibians, and relates the experimental data to the genesis of the epibulbar cyst. On the basis of the experimental data in low vertebrates and invertebrates, the author concludes that it is very probable that the human optic vesicle has the capacity of autodifferentiation and organogenesis. (20 figures, 23 references) Ray K. Daily.

Wagner, Friedrich. **Bilateral coloboma of the lens in arachnodactyly.** Klin. Monatsbl. f. Augenh. **120**:640-643, 1952.

This rare combination of anomalies in a 40-year-old man is assumed to be an expression of the status dysraphicus (Passow). (2 figures, 15 references) Frederick C. Blodi.

20

HYGIENE, SOCIOLOGY, EDUCATION, AND HISTORY

Biernacka-Biesiekierska, Jadwiga. **Pathogenesis of eye conditions caused by dust.** Klinika Oczna **22**:89-102, 1952.

The presence of dust in the air (particles less than 0.05 mm. in size are classified as dust) is characteristic of many types of industries and presents definite hazard to the health of workers and their eyes. Dust may be active chemically or be inert. Many parts of the eye may be affected. In the lids there may be permanent discoloration and local areas of inflammation with ulceration, entropion, ectropion, trichiasis, and blepharophimosis. The conjunctiva reacts with congestion and hyperplasia forming pinguecula, pterygium, and granulation tissue. Allergic conjunctivitis is quite frequent. Dust particles affect mainly the exposed part of the cornea. Pain, lacrimation and photophobia are present in the beginning. Minor abrasions and scars can be seen with the slitlamp. In chronic irritation there is lowering of the corneal sensitivity. Superficial scars with loss of the Bowman's membrane and degeneration of the corneal stroma may result. Chronic iritation by the dust may cause gradual atrophy of the tear gland and chronic inflammation of the lacrimal duct. Different types of individual occupations are described where dust irritation is prevalent. The author investigated the production of washing powder. The chemical and physical composition was determined and all phases of the production were checked. Only some of these phases caused the formation of dust. He also investigated conditions of work in sandstone quaries. In some phases of production there was a great deal of dust in the air. Because of involvement of the respiratory tract, different types of masks and goggles were advised, depending on the type of work. Investigation of the density of particles in the dusty air was conducted. Safety limits must be worked out for each type of industry. Investigation of the size of dust particles demonstrated that the most damaging chemically are the particles below five microns in size. The larger particles irritate tissues mechanically. (2 figures, 2 tables) Sylvan Brandon.

Dale, Henry. **The eye as a physiological reagent. (Bowman Lecture).** Tr. Ophth. Soc. U. Kingdom **71**:117-132, 1951.

The author emphasizes the value of the eye in physiologic research, which was used so dramatically by Bowman in some of the questions he propounded before the technical help which is available now was known. The observations have been continued and now at the Institute of Ophthalmology reactions of the eye and of its extrinsic and intrinsic musculature and their nerve supply are studied.

Beulah Cushman.

Jaensch, P. A. **The importance of sightsaving schools.** Klin. Monatsbl. f. Augenh. **121**:1-9, 1952.

Germany has only three sightsaving schools (compared with 653 in the USA). The schools are of utmost importance for visually handicapped children. For admission the vision must be better than 0.04 and worse than 0.25 and the children should be mentally normal. It is the greatest disadvantage to these children to be sent to schools for the blind or mentally defective. The financial problems of such schools are discussed.

Frederick C. Blodi.

Ruedemann, A. D., and Peterson, A. K. **Eye health evaluation and maintenance.** Arch. Indust. Hyg. **6**:243-251, Sept., 1952.

Ruedemann feels that success in learning in schools and quantitative and qualitative productivity of a worker in industry depend to a great extent on foveal coordination. This coordination is difficult or impossible if muscle defects are present. Fatigue from refractive errors may play a part. In the student the eyes work harder at the expense and sacrifice of the memory functions. In industry these ocular abnormalities bring early fatigue and therefore errors in judgment. Normal visual acuity (20/20) alone is unimportant if hyperopia or muscle imbalances are also present. Ocular difficulties should be discovered and reeducation begun in the first four or five years of life.

Peterson accentuates the need for community responsibility for eye health and Ruedemann believes that this should come early in life.

Industry has availed itself of testing by special methods such as the ortho-rater and has in many instances abandoned the use of eye-testing charts. Attention to improper illumination and color deficiencies has been an important concern of the larger plants. Periodic examinations of regular employees have become an important procedure in the maintenance of the health of the eyes.

F. M. Crage.

Spaeth, Edmund B. **Senility and senescence in ophthalmology.** Rocky Mountain M. J. **49**:506-513, June, 1952.

The histologic changes found by Kornsweig in the postmortem study of 120 eyes obtained from patients who had died at an age between 70 and 90 years are listed. Those changes which are significant in the degenerative process of old age are discussed in greater detail. Senile changes in the blood vessels are responsible for many pathologic conditions; choroidal sclerosis causes senile macular degeneration and senile peripapillary halo. Indirectly, vascular changes bring about noninflammatory forms of chronic simple glaucoma by sclerosis of the vessels draining Schlemm's canal, retinopathies of arteriosclerosis, diabetes, nephritis and others. Senile cystic degeneration at the periphery will predispose an individual to retinal detachment and centrally is the forerunner of the macular holes. Fibrosis and hyalinization of the ciliary muscle and the suspensory ligament of the lens along with loss of elasticity of the lens causes presbyopia. The pathogenesis and pathology of senile cataract is discussed.

James V. Bolger.

NEWS ITEMS

Edited by Donald J. Lyle, M.D.
601 Union Trust Building, Cincinnati 2

News items should reach the editor by the 12th of the month but, to receive adequate publicity, notices of postgraduate courses, meetings, and so forth should be received at least three months before the date of occurrence.

Deaths

Dr. Frederick John Hoitash, Huntington, West Virginia, died November 29, 1952, aged 69 years.

Announcements

Texas residencies open

Three residencies in ophthalmology will be open this summer in Houston, Texas. A basic course is required. For further information, please communicate with Dr. E. L. Goar, 1304 Walker Avenue, Houston 2, Texas.

Meeting at Walter Reed

A spring meeting on "Recent advances in ophthalmology," will be held at Walter Reed Army Medical Center on May 21st and 22nd. Speakers will include: Prof. A. Franceschetti, Geneva, Switzerland; Dr. Alston Callahan, Birmingham; Dr. Michael J. Hogan, San Francisco; Dr. Irving H. Leopold, Philadelphia; Dr. Frank B. Walsh, Baltimore; and Mrs. Helenor C. Wilder, Washington, D.C. Col. H. Amory, Col. J. H. King, Jr., and Col. A. Lowrey will also lecture.

Interested civilian physicians are invited to attend. There is no fee but applications should be made by April 21, 1953, to the Commanding General, Walter Reed Army Medical Center, Washington, D.C. Attention: Col. J. H. King, Jr., director, Ophthalmology Course.

Ophthalmologists needed

The New York City Department of Health has openings for ophthalmologists in its diagnostic eye clinic, 22 of which are operated for patients up to 21 years of age throughout the city. These clinics provide diagnostic service only. Each ophthalmologist is expected to see about 12 children per three-hour clinic session.

An applicant must (1) either be a diplomate of the American Board of Ophthalmology or have completed a residency in ophthalmology in a hospital approved for residency training by the A. M. A. Council on Medical Education and Hospitals and be eligible to take the examination of the American Board of Ophthalmology and (2) be licensed to practice medicine in New York.

Clinic ophthalmologists are paid $11.30 per three hour clinic session, are given a prorated amount of annual vacation with salary, and may join the New York City Retirement System. Application should be made in writing to Dr. Helen M. Wallace, director, Bureau for Handicapped Children.

Placement service

The American Association of Orthoptic Technicians wishes to announce that a confidential placement service has been organized for positions for certified orthoptic technicians. Information regarding available technicians and positions open can be received by writing Mrs. Louisa Wells Kramer, 1779 Massachusetts Avenue, N.W., Washington 6, D.C.

Neurology courses

The American Academy of Neurology is offering special courses in various aspects of neurology and allied disciplines on April 6, 7, and 8, 1953, at the Edgewater Beach Hotel, Chicago, just preceding the fifth annual meeting of the academy. The courses in "Neuropathology," "Clinical electro-encephalography," and "Neuroroentgenology," are being offered again this year. In addition, there will be courses on "Special problems in clinical electroencephalography," "Episodic disturbances of the nervous system," "Clinical neuro-ophthalmology," "Language disabilities," "Brain tumors," and "Neurologic anatomy."

For further information, write Mrs. J. C. McKinley, 19 Millard Hall, University of Minnesota, Minneapolis, Minnesota.

Orthoptic examinations

The annual examination of orthoptic technicians by the American Orthoptic Council will be conducted in August and October, 1953.

The written examination will be nonassembled and will take place on Thursday, August 27, in certain assigned cities and offices and will be proctored by designated ophthalmologists.

The oral and practical examinations will be on Saturday, October 10th, in Chicago just preceding the meeting of the American Academy of Ophthalmology and Otolaryngology.

Application for examination will be received by the office of the secretary of the American Orthoptic Council, Dr. Frank D. Costenbader, 1605 22nd Street, N.W., Washington 8, D.C., and must be accompanied by the examination fee of $30.00. Applications will not be accepted after July 1, 1953.

Miscellaneous

Texas research grants

During the past year, the Ophthalmic Research Foundation of Texas has given three grants of $1,000.00 each for research in ophthalmology to three medical schools in Texas, Texas University-Main Branch, Baylor University, and Southwestern University.

Chicago clinical conference

Three symposia were held during the recent three-day clinical conference sponsored by the Chicago Ophthalmological Society and held at the Sheraton Hotel, Chicago.

Dr. William F. Hughes, Jr., Chicago, was moderator for the symposium on "Retrolental fibroplasia." Dr. William C. Owens, Baltimore, discussed "Clinical appearance and pathology"; Dr. Milton Scheffler, Chicago, "Differential diagnosis"; Dr. Walter R. Hepner, Galveston, "Pediatric aspects"; Dr. V. Everett Kinsey, Detroit, "Experimental studies"; and Dr. Arlington C. Krause, Chicago, "Experiences with therapy."

For the symposium on "Recent advances in ophthalmic surgery," Dr. Frank W. Newell, Chicago, was moderator. "Local anesthesia," was discussed by Dr. Daniel Snydacker, Chicago; "General anesthesia," Dr. Harold Harris, Evanston; "Ocular muscles," Dr. Martin J. Urist, Benton Harbor; "Keratoplasty," Dr. William F. Hughes, Jr., Chicago; "Retinal detachment," Dr. Peter C. Kronfeld, Chicago; "Glaucoma," Dr. Joseph S. Haas, Chicago; "Cataract," Dr. Derrick Vail, Chicago.

Dr. Joseph S. Haas, Chicago, acted as moderator for the symposium on "The aqueous humor." Dr. Georgiana D. Theobald, Oak Park, Illinois, discussed "The canal of Schlemm"; Dr. V. Everett Kinsey, Detroit, "The formation of aqueous"; and Dr. Bernard Becker, Baltimore, "Tonography and the drainage of aqueous."

Surgical clinics were held by Dr. J. Robert Fitzgerald, Dr. William F. Hughes, Jr., Dr. T. N. Zekman, Dr. Derrick Vail, Dr. J. M. Donegan, Dr. Peter C. Kronfeld, and Dr. J. W. Clark. Papers were presented by Dr. Bernard Becker on "Recent studies concerning the pathogenesis of diabetic retinopathy"; Dr. Frank B. Walsh, Baltimore, "Myasthenia gravis"; and Dr. William C. Owens, "Accommodative dysharmonic esotropia." Dr. Walsh gave the Sanford R. Gifford Memorial Lecture at the close of the conference. The subject of his address was "Some aspects of calcification in ophthalmology."

Oxford congress

The Oxford Ophthalmological Congress will assemble at Balliol College, Broad Street, Oxford, England, on the evening of Sunday, July 5, 1953, and meetings will be held on July 6th, 7th, and 8th in the lecture hall of the Sir William Dunn School of Pathology, South Park Road.

Among the subjects to be discussed are "Visual requirements in relation to modern travel." The introducers will be Sir Stewart Duke-Elder, London; Air Commodore J. C. Neely, R. A. F. and London; and Prof. W. J. B. Riddell, Glasgow. The discussion on "Ophthalmic nursing" will be opened by Dr. John Marshall, Glasgow, Mr. Derek Ainslie, London, and Miss MacKellar, Moorfields Hospital, London. Mr. Keith Lyle, London, will deliver the Doyne Memorial Lecture on the morning of July 7th.

Gill hospital congress

Ophthalmic speakers at the 26th annual spring congress of the Gill Memorial Eye, Ear and Throat Hospital, Roanoke, Virginia, April 6th to 11th will be:

Dr. Glen G. Gibson, Philadelphia, who will speak on "The differential diagnosis of retinopathy," "Classification of retinal arteriolar signs of hypertension," and "Retinal detachments." Mr. H. B. Stallard, London, England, "The irradiation treatment of retinoblastoma," "Anterior flap sclerotomy with basal iridencleisis," "Lamellar scleral resection," and "Some technical points in strabismus operations."

Dr. A. B. Reese, New York, will speak on "Cataract extraction," and "The present concept of primary glaucoma." Dr. Irving H. Leopold, Philadelphia, "Principles of autonomic drug therapy in ophthalmology," "Present status of antibiotics in ophthalmology," "Therapy of diabetic retinopathy and other vascular fundus disorders," "Medical therapy of glaucoma," and "Application of steroids in ocular diseases."

Dr. Arthur Linksz, New York, will discuss "Visual acuity," "Astigmatic charts, cross cylinder, cylinder retinoscopy," "Cycloplegic vs. noncycloplegic refraction," and "Refractive errors and space perception."

Dr. Raymond Meek, New York, "Surgery of the commissures," "The Elliot trephining operation vs. iridencleisis in glaucoma," "The management of eye injuries," and "Orbital implants."

Dr. Rudolf Aebli, New York, "The nonoperative management of heterophorias and heterotropias," "Congenital anomalies: Adhesion syndromes," "Practical considerations in ocular muscle surgery," and "The relationship of pseudoptosis to muscle tropias and the palpebral aperture." Dr. Milton Berliner, New York, "Series of four lectures on biomicroscopy: Technique; Conjunctiva, cornea, and iris; Lens examination; Vitreous and fundus examination."

Dr. Jack S. Guyton, Baltimore, "Muscle surgery." Dr. Charles E. Iliff, Baltimore, "Simplified dacryocystorhinostomy," and "Treatment of limbal lesions."

University of Toronto course

Guest surgeons at the eye surgery refresher course of the University of Toronto Faculty of Medicine, to be held in Toronto April 13th through 17th will be Dr. D. K. Pischel, San Francisco, and Mr. H. B. Stallard, F.R.C.S., London, England.

On the program are: "Pathogenesis of retinal detachment," "The clinical examination of the vitreous," "Diagnosis and differential diagnosis of retinal detachment," "Operative treatment of retinal detachment," "Complications of operative treatment of retinal detachment," and "Minor surgical procedures," by Dr. Pischel.

"Retinoblastoma treated with radium," "Anterior flap sclerotomy with basal iridencleisis," "Surgery of entropion and ectropion," "Some technical points in strabismus surgery," "Intraocular foreign body sur-

gery," and "Dacryocystorhinostomy," by Mr. Stallard.

In addition, there will be surgical demonstrations by Dr. Pischel and Mr. Stallard, as well as lectures and surgical demonstrations by the following:

Dr. H. L. Ormsby, Dr. J. C. McCulloch, Dr. A. J. Elliot, Dr. A. E. MacDonald, Dr. R. G. C. Kelly, Dr. T. H. Hodgson, Dr. H. M. Macrae, Dr. J. C. Hill, Dr. A. L. Morgan, Dr. J. S. Crawford, Dr. W. W. Wright, Dr. J. F. A. Johnston, Dr. O. B. Richardson, and Dr. W. R. F. Luke.

Societies

Charlotte society formed

Meetings of the newly formed Charlotte (North Carolina) Ophthalmological Society will be held bimonthly. Charter members of the society include: Dr. H. C. Neblett, Dr. C. B. Foster, Dr. W. R. Graham, Dr. T. D. Ghent, Dr. H. T. Holden, Dr. M. J. Hough, Dr. Ruth Leonard, Dr. M. J. Lymberis, Dr. F. C. Smith, and Dr. J. D. Stratton. Officers elected were: President, Dr. H. C. Neblett; vice president, Dr. J. D. Stratton; secretary-treasurer, Dr. T. D. Ghent.

Richmond officers

Newly elected officers of the Richmond (Virginia) Eye, Ear, Nose and Throat Society are: President, Dr. J. Warren Montague; secretary-treasurer, Dr. Charles N. Romaine. Meetings are held the first Tuesday of January, March, May and October, at the Commonwealth Club.

Puget sound academy

Officers for 1953 of the Puget Sound Academy of Ophthalmology and Otolaryngology are: President, Dr. Clifton E. Benson, Bremerton, Washington; president elect, Dr. Carl D. F. Jensen, Seattle; secretary, Dr. Willard F. Goff, Seattle.

Nassau meetings

At the February 23rd meeting of the Nassau Ophthalmological Society held at the Flagstone, Hempstead, New York, Dr. Paul Chandler, Boston, spoke on "Narrow-angle glaucoma." Dr. Conrad Berens opened the discussion. Meetings of the society for 1953 are scheduled for April 27th, September 28th, and November 23rd. Officers are: President, Dr. Wendell L. Hughes; vice president, Dr. Arthur A. Scimeca; secretary-treasurer, Dr. Ward L. Mould; assistant secretary-treasurer, Dr. Eugene T. Buckley; members of the council, Dr. Milton T. Gaillard and Dr. A. Milton Goldman.

Reading program

At the 131st regular meeting of the Reading (Pennsylvania) Eye, Ear, Nose and Throat Society, Dr. John T. Dickinson, Pittsburgh, spoke on "Primary and delayed repair of traumatic defects of the face." Preceding the regular meeting a study club meeting was conducted on the subject of "Modern treatment of uveitis," with Dr. John M. Wotring, Reading, acting as moderator, and Dr. Harold L. Strause, Reading, instructor.

1953 North and South Carolina meeting

Arrangements have been completed for a joint meeting of the South Carolina Society of Ophthalmology and Otolaryngology and the North Carolina Eye, Ear, Nose and Throat Society to be held at the Francis Marion Hotel, Charleston, South Carolina, on September 14, 15, and 16, 1953. Otolaryngologists who will take part in the program are Dr. Wayne Slaughter, Chicago; Dr. John R. Lindsay, Chicago; and Dr. Theodore Walsh, Saint Louis. Ophthalmologists participating will be Dr. Derrick Vail, Chicago; Dr. David G. Cogan, Boston; and Dr. R. Townley Paton, New York.

There will be a panel discussion on vertigo from the ophthalmic, otolaryngologic, neurologic, and internal medical standpoints. Dr. Charles Kunkle of Duke University will be the guest neurologist and Dr. Vince Mosely of the Medical College of South Carolina will be the guest internist.

For further information communicate with: Dr. George T. Ferguson, McPherson Hospital, Durham, North Carolina, or Dr. Roderick Macdonald, 330 East Main Street, Rock Hill, South Carolina.

Course on glaucoma

A course on "Glaucoma," with particular emphasis on gonioscopy and study of the anterior angle will be given at the Brooklyn Eye and Ear Hospital on May 4, 5, and 6, 1953. Ample opportunity for practical instruction in the use of the goniolens will be given and material from the Glaucoma Clinic will be utilized. One afternoon will be devoted to surgical indications and technique of glaucoma surgery in the operating room.

The faculty will include Dr. E. Clifford Place, Dr. Walter V. Moore, Dr. M. A. Lasky, Dr. Daniel Kravitz, and Dr. Arthur Shainhouse.

Application and the fee of $40.00 for registration by ophthalmologists may be addressed to Dr. Daniel Kravitz, Brooklyn Eye and Ear Hospital, 29 Greene Avenue, Brooklyn 38, New York.

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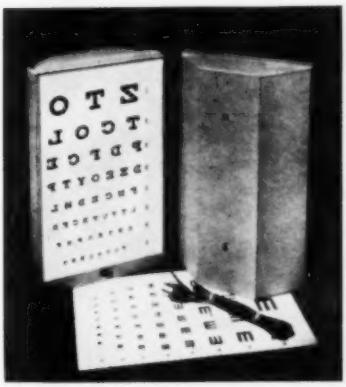

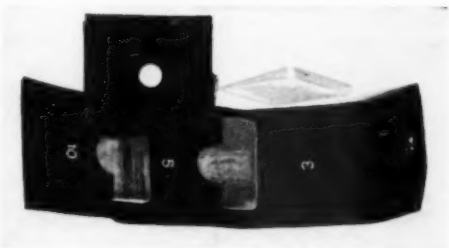

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Recently published, this booklet contains much interesting material relating to AO Monoplex Plastic Artificial Eyes and AO Implants. The Eye section of the booklet describes Monoplex stock eyes, modifications possible with these premade prostheses, and custom-made eyes. Three types of Orbital Implants are discussed: Integrated and Buried Mesh types, and Spheres.

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